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VOL. II.—42ND YEAR SYDNEY, SATURDAY, SEPTEMBER 3, 1955 No. 10

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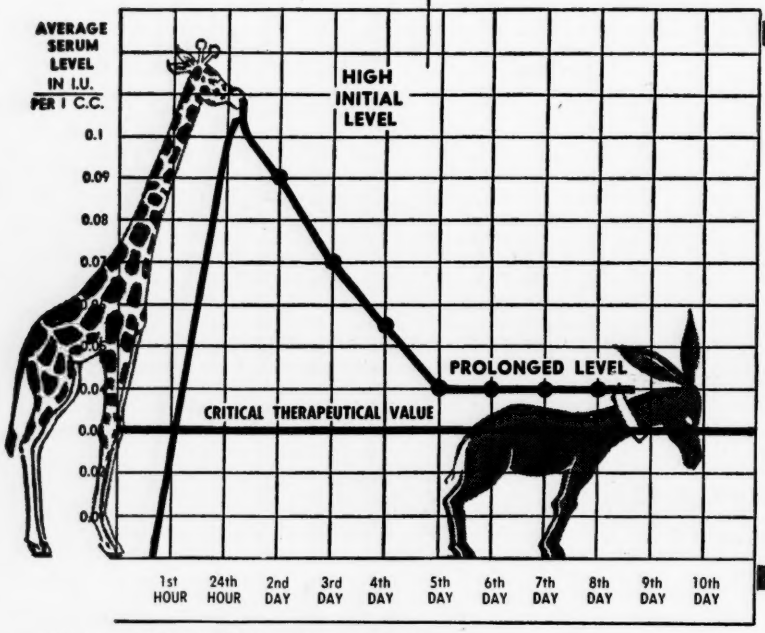

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A MEDICAL HISTORY OF THE WOLLONGONG DISTRICT.

By H. H. LEE, M.B., Ch.M., F.R.A.C.S.,
Wollongong.

Kiama.

It is obviously impossible for me in the time at my disposal to give a pen picture of all the men who have been at Kiama in the last ninety years, so I fear that my remarks may somewhat resemble the Book of Genesis.

The first recorded name is that of Dr. Menzies (1860). He combined farming with medical practice. I have no record of how he divided his time. His deeds are shrouded in the mists of antiquity. He was succeeded by Dr. Taylor and Dr. Nolan. They were constantly at loggerheads and their squabbles were fully recorded in the *Kiama Independent* (1865).

In 1870 two brothers, Dr. Herman Tarrent and Dr. William Tarrent, practised together. Dr. William Tarrent was killed in a buggy accident when driving home from a Masonic meeting. His horses bolted, and they, or he, capsized the buggy. Dr. Herman Tarrent became the Member of the Legislative Assembly for the district and went to live in Sydney. He was later elected Worshipful Grand Master of the New South Wales Lodge of Freemasons. He must have been a man of very considerable ability. When his term of office expired he went to England and returned with secret cures for every known disease. He started as a medical specialist (I presume of everything) and ran a large medical institute in Macquarie Street. He advertised in all the papers and had a large photograph of himself in *The Sydney Morning Herald.*

Dr. Caird and Dr. Lacey succeeded Dr. William Tarrent. Dr. Caird died soon of what is described as heart disease, and Dr. Lacey was killed by a falling tree. Dr. Calib Terry succeeded him, and years later Dr. Hedley Terry joined him. I remember him best as an excellent left-handed batsman. I was captain of Wollongong Club in those days, so his memory is green.

Dr. Fox carried on from 1905; he introduced the first motor-car—as Robertson and Kangaroo Valley were in his practice, he certainly needed one.

Dr. Fletcher took over in 1929, but after a time he went to Sydney to do radiography. Dr. Fletcher was succeeded by Dr. B. A. Stephen, who, in his own words, is struggling on. Judging from his appearance, I think the struggle has been a successful one.

Dr. Robert Beith went to Kiama in 1899 and practised there for twenty-six years. When he left, a tumultuous valedictory meeting was held in his honour. He was a

Read at the fourth annual post-graduate course of the New South Wales Federation of Country Local Associations, Wollongong, March 14 to 18, 1955.

dour Scotchman. Dr. Corner succeeded Dr. Beith; he left to specialize in ear, nose and throat work. Dr. John Harris succeeded him; but he, whilst attending a ball, died suddenly of coronary occlusion. Kiama doctors seemed to be addicted to sudden and violent deaths.

Dr. Max Cramm came next, and the two practices were united for the first time.

One other man I must mention, though I do not know where he fits in. He was known as "Leather-Bag" Reid. He was Dr. George Reid, and he was in Kiama about 1890. A tall, dignified, handsome man, he always did his rounds on foot, wearing a top hat and morning suit. I think he must have found the humid atmosphere trying, but anyhow he kept up the dignity of the profession.

Acknowledgement.

I am indebted to Dr. B. A. Stephen and the *Kiama Independent* for most of my information.

Albion Park.

Albion Park is a small town on the South Coast road between Kiama and Dapto. Its population is entirely agricultural. It was for many years a sporting rendezvous for Sydney visitors. Lake Illawarra was about half a mile away, with good fishing, and the country carried plenty of quail in good seasons. Jack Raftery, of Raftery's Hotel, kept a motor-launch, and pointer and setter dogs. His hotel was rarely empty. To the best of my recollection, for many years only two men attempted to practise at Albion Park—Dr. Vanzetti, whose deeds are lost in the mists of antiquity, and Dr. Bateman. Dr. Bateman was there for many years. He built himself a very nice house, "Revensworth". He was an elderly man with a family of eight. His eldest son was drowned in Lake Illawarra when duck-shooting. His second son, with whom I had much good quail-shooting (I was an undergraduate then), inherited an estate in England that had been in the family since the thirteenth century. His third son graduated with first-class honours at the University of Sydney and has been practising at Windsor. Dr. Bateman himself was a delicate man, a hæmophiliac. He was extremely well educated and very interested in Volapük. This was to be a universal world language which was to assist in abolishing war. Dr. Bateman was an enthusiastic disciple, but, I fear, met with little support. He once embarrassed me considerably. As I was walking off the court after a tennis match he approached me with an open "Virgil" with a heavily underlined passage and asked me if I would oblige him with my rendering of it. I was doing arts at that time at the University, so I suppose he thought "Virgil" would be an open book to me. It was not.

For many years Albion Park had no resident doctor; now I understand it has two—Dr. O'Dea and Dr. Needham.

Dapto.

In the dim and distant past Dapto was a sparsely populated, scattered farming and grazing district. Wheat, butter and cheese were the chief reasons for its existence. A small sailing ship, the *Dairy Maid*, used to call at Wollongong and Shell Harbour at irregular intervals to convey these products to Sydney. The inhabitants of Dapto received medical attention from the Kiama and Wollongong doctors. Dr. Curgenven settled there some time in the late eighties. I have meagre accounts of him, but I fear he met with financial difficulty. The wheat industry had been ruined by rust and the town had only the dairy industry to rely on. By this time the railway had reached Dapto and the farmers were able to send their products away easily and also were able themselves to reach larger towns. When I last heard of Dr. Curgenven he was laying the odds at Randwick. Let us hope he was luckier as a fielder than as a medical practitioner.

For some years Dapto had no doctor; the Wollongong men worked it for what it was worth. As none of us had motor-cars, and as the farms were very scattered, it was a full-sized man's job. Some years later the Dapto Smelting Works started on the edge of the lake and the town began to flourish. Dr. Scott commenced practice there. I qualified soon after; he and I did a lot of work together. He was a very good practitioner when at his best, and a very nice man. Before he came to Dapto he practised at Narrabri. My brother-in-law, Maurice Barton, had a station thereby. They met in an hotel after a day's racing. They discussed the merits of William Tell and the apple. Barton, who was an excellent rifle shot, offered to shoot a cork off Scott's head. Scott was willing, so they went outside and Barton blazed away at the cork with a 32 "Winchester". All would have been well but for the fact that (owing, I suppose, to the hot weather) Barton had omitted to readjust his sights for the required distance. He blew the cork to pieces, but a neat tuft of hair floated into the air and a thin trickle of blood ran down Scott's cheek. However, no one was really hurt, and after that I understand a good time was had by all.

Whilst in Dapto Dr. Scott achieved fame in two ways. He was the first man to bring into the district a motor-buggy. These were terrible machines. They were high double buggies with an engine under the seat, transmission by two belts, solid tyres, and top speed of about twelve miles an hour. They made a noise like a traction engine, gave out a terrible smell, and never did more than twenty miles without requiring some adjustment. Dr. Scott's second claim to immortality was that he was, as far as I know, the only doctor in Australia ever to be attacked by a man-eating tiger. He was walking back from a farm-house on Farmborough Road when he was seized from behind by a large animal that he took to be a tiger. The tiger grabbed him by the waist and proceeded to carry him to the road, I presume to eat him on the main road. Dragging him through a barbed-wire fence, he broke a bottle of chloroform that Scott had in his pocket. The smell presumably alarmed the beast, for he dropped Scott and made off. He has never been seen since. Scott escaped but was very badly scratched. He told me the story himself.

In the course of time the Dapto Smelting Works failed; they were moved to Port Kembla, but went finally broke there. Dapto then returned to peaceful slumber. Some time later Dr. Henry practised there. He was an old man considerably past his best. He was one of the first four medical graduates of the University of Sydney (Critchley-Hinder, Mills, Henry and one other). Compare that with the number graduating each year now. Perhaps I may be excused for saying that I was the first Wollongong man to take the degree of bachelor of medicine of the University of Sydney. With the advent of the iron and steel works at Port Kembla and several new coal mines in the district, Dapto again sprang into life, and it is now a flourishing town. In the last twenty years or so a stream of medical men have passed through Dapto. Few have stayed long, for though the practice is quite lucrative it has no outlet and leads nowhere.

Bulli.

Bulli is the second largest town in the area. Dr. Sturt was, I think, the first man to practise there. He was an old man; in fact, I thought him a very old man, but time has made me more broad-minded. He retired in 1889 and handed his practice to his brother, a Dr. Clifton Sturt. The latter in turn transferred it to Dr. James Thompson. At this time there were only three collieries in the district—Mount Keira, Mount Pleasant, and Old Bulli—but fresh collieries were opening up—South Bulli, Clifton, Mount Kembla, Mount Pleasant and Victoria. My father was medical officer to many of them and he had a huge practice extending from Albion Park to Clifton. As there were no motor-cars in those days, his requirements in horseflesh were considerable. Finding the work too heavy, he built a house at Bulli and installed an assistant, Dr. Naegal, and that was the beginning of the second practice in Bulli. Dr. Naegel was succeeded by Dr. Blackall, who after a time left to go to Queanbeyan, where he practised till he retired. He was many times mayor of Queanbeyan.

In 1890 my father sold his interest in Bulli to Dr. Kane. In 1897 Dr. Cummins succeded Dr. Kane, and Dr. O'Connor succeeded Dr. Cummins. Dr. O'Connor sold the practice to Dr. Palmer, who is still with us; he shares with me the honour of being the only original members of the South-Eastern Medical Association. Dr. Palmer started in 1890. Dr. Clifton Sturt, who succeeded his brother in the original

Bulli practice, sold it to Dr. Dixon. Dr. Dixon had a very large practice extending as far as Stanwell Park; he had an assistant, Dr. Steel, to whom he eventually sold the Clifton end of his practice. He in turn sold it to Dr. Goldie. Dr. Goldie sold it to Dr. Fetherstone when he went to World War I, and on his return Dr. Goldie bought a practice at Corrimal. He later bought Dr. Kerr's practice in Wollongong, where he died quite recently.

Part of Dr. Dixon's practice was bought by Dr. Macpherson, who sold his interest to Dr. Crosslé when he went to World War I. Dr. Crosslé, after some years of very successful practice, transferred his interest to Dr. Cook, who is still with us.

In Corrimal, the adjoining practice, there have been innumerable changes. Dr. Park, Dr. Roper, Dr. Caselberg and Dr. Diment have stayed longest. There were others, but they stayed only a short time. I can make a personal reference to only four—three from Bulli and one from Corrimal. First and foremost comes Charles Palmer. He practised for forty-three years in Bulli, and any man who can practise for as long as that in an industrial town and get away with his life deserves congratulations. Dr. Palmer served with the Australian Imperial Force in the first World War. His son, Dr. E. Palmer, served in the second World War, and his two daughters as nursing sisters served with distinction in the second World War right through the siege of Malta. If there is an Australian family with a better war record than that, I have not heard of it.

Dr. Crosslé I would describe as a polished Irish gentleman—an extremely nice man. He became a brilliant surgeon and did excellent work in the Bulli-Thirroul-Clifton area. He left to go to Macquarie Street, where he died after a few years.

Dr. Cook, our former president, most of you know; he has done and is still doing excellent work as a practitioner and as our representative on the Council of the New South Wales Branch of the British Medical Association.

Dr. Diment, of Corrimal, began in this district as a house surgeon in the Wollongong Hospital. I had the pleasure of working with him for six months. He was always punctual, always helpful, and in the operating theatre never gave advice unless it was asked for—a model house surgeon.

Port Kembla.

Port Kembla, four miles south of Wollongong, has made phenomenal growth in the last fifty years. When I first knew it it had only two inhabitants—Mr. Downie and Mr. Sinclair. I would not hazard a guess as to its population now; perhaps it is 10,000. When the electrolytic works were built there progress began. They were followed by the Metal Manufactures, the iron and steel works, and then Lysaght's works. For many years Dr. Kerr and I shared the Port Kembla work, but after the end of World War I I built a house there and installed an assistant, Dr. Kirkwood. In three years' time I moved him into Wollongong, putting Dr. Geeves in his place. In three years again Dr. Perkins replaced Dr. Geeves. In three years again came the financial depression and I withdrew my man, as I could not afford to keep him there. After a hiatus of about two years Dr. McKellar started there. After a short stay he sold the practice to Dr. Luscomb, who with various partners and assistants still holds it.

Wollongong: The Queen City of the South.

The first record I have of a medical man in Wollongong is that of Dr. Redall. He and his brother came together. The brother was a farmer living on the edge of Lake Illawarra, who gave his name to Redall's Bay, and those of you who fish may know Redall Reef, an excellent place for bream and blackfish. Dr. Redall was part farmer and part doctor; I do not know how his time was divided. The next man was Dr. Thomas, who practised in Wollongong in a house still standing opposite the Church of England. He was followed by Dr. Marshall; he practised in the house in which I now have my professional rooms; he married a Wollongong girl, and between them they gave the name of Marshall Mount to a district between Dapto and Albion Park. The Osborne family built there a glorious house known as Marshall Mount House; it is still standing, but is only a shadow of its former greatness. In course of time Dr. Marshall went, I believe, to Macquarie Street, and Dr. Thompson succeeded him. He was a retired army doctor, and he lived opposite the Court House. He sent his only son home to qualify in medicine. The son did so, but died of pneumonia almost as soon as he returned. He and his father and mother are all buried in the Red Church graveyard (Osborne Memorial Church).

Dr. Gould was practising in opposition to Dr. Thompson; he had a very small connexion. From him my father bought his practice. As he gave only £20 for it, it must have been a very small connexion; this was in 1884. My father, young, energetic and, incidentally, an extremely competent man, soon established himself. Wollongong had 800 inhabitants, though the district was large. The railway from Sydney to Wollongong was begun soon after my father and family settled in the town. I hasten to add that I was a very small boy then. Up till then all communication with Sydney had been by paddle steamer or by coach that ran twice a week to Campbelltown, where passengers picked up the train. The coach, a five-horse one, started from the Queen's Hotel, now the Queen's Flats. My father had the contract to attend to all the workers on the line from Wollongong to Otford. He had to ride the line twice a week. It was a very lucrative job, but a very hard one. It was saddle work all the time. He was, happily, an excellent horseman and could get more out of a horse than any man I ever saw who was not a professional jockey. At one time we had thirteen horses in the paddock—eleven for his own riding, my mother's hack and my little pony. Thirty odd years later, Dr. Kirkwood, my then partner, and I had the same contract to ride the line from the top of the Macquarie Pass to Unanderra, a 33-mile ride over the roughest possible country. We both enjoyed it; it seemed to be a real man's work. It confirmed my father's opinion that the pick-and-shovel "dinkum Aussies" were the best men in the world to work for. At the time of the Sydney to Wollongong railway, Wollongong had a small hospital of 12 beds, the Albert Memorial Hospital, staffed by a married couple. The building still remains. The hospital was always full; typhoid fever was endemic, as there was no water supply and no sewerage. The Wollongong District Hospital was opened in 1908. It has been constantly added to, but is still inadequate for the demands made on it. As the town grew, Dr. Fielder settled here, and in course of time he sold out to Dr. Kerr. Dr. Thompson died, and Dr. Jarvie Hood succeeded him. His brother was the most fashionable physician in Sydney at that time. He was a most attractive man, and had he lived I think my father and he would have gone into partnership, but unfortunately he died very young. When Dr. Jarvie Hood died, Dr. Thomas Wade filled his place. He in turn transferred the practice to Dr. W. L. Kirkwood, who sold it to Dr. Jackson when Dr. Kirkwood went off to World War I. In due course Dr. Jackson transferred the practice to Dr. Street, who happily is still with us. In 1902, I, having just returned from a year's post-graduate work in England, joined my father, William Kerr joined his brother, and the three practices—Lee, Kerr and Wade—consisting of five men, held the district for many years. My father retired in 1911, leaving me in charge. I worked the practice sometimes with a partner, sometimes with an assistant and sometimes with both, till 1954, when I resigned all my lodge and contract appointments; I am now doing only consulting work. For sentimental reasons I did not sell my practice; I simply abandoned it. The Kerrs sold their practice to Dr. Goldie, who most unfortunately died young. Dr. Finlayson, Dr. Wiseman and Dr. Rich now control it. Dr. Street, Dr. Suttor and Dr. Davis hold the old Wade practice.

During the last years several new practices have started. Dr. Duck, with Dr. Walton as his partner, Dr. Bedcomber, Dr. McKinnon as I have already mentioned moved in from Dapto, Dr. James, Dr. M. and K. B. Karn, Dr. Outhred, Dr. Fraser, Dr. Diment, Dr. Powrie and Dr. Pittar are situated in what you might describe as the suburbs of Wollongong. There are also specialists on every part of

the body known to man or women; they are too numerous for me to include them. If you compare this list with the two men who practised here when my father began here seventy-one years ago you will get some idea of how the district has grown.

In the fifty-odd years during which I have practised in this district, I have worked with many men. Three men stand out as men who have left their "footprints on the sands of time": first, my father, who from nothing established one of the largest country practices in New South Wales; Dr. John Kerr, a shrewd, dour Scotchman, whose work was excellent; and Dr. Francis Crosslé, of Bulli, a man who became a brilliant surgeon and did excellent work in the Bulli-Thirroul-Clifton area. God rest their souls.

AN ADDRESS.[1]

By B. A. COOK, M.B., Ch.M.,
President, Federation of Country Local Associations of New South Wales.

THE Federation of Country Local Associations, hereinafter called "FOCLA", has decided that each year at the beginning of the annual post-graduate course an oration will be delivered in memory of a prominent country general practitioner who has been translated to his eternal reward after completion of his life's work. This year's oration has been dedicated to the memory of the late Dr. Roy Dadson Mulvey, of Bathurst.

Roy Dadson Mulvey was born at Newcastle, New South Wales. He attended the Cook's Hill High School and the Maitland Boys' High School, and at sixteen entered the Teachers' College, meanwhile attending the University, and graduated Bachelor of Arts in 1912, and Bachelor of Science in 1914. His teaching career culminated in his being granted a travelling scholarship in 1914. But there was a sterner travelling assignment, he felt, to be undertaken, for he enlisted for service overseas, after the death of a brother at the Dardanelles. He rose rapidly in the ranks to the command of the Eighth Trench Mortar Battery, gaining the Military Cross, and having been mentioned in dispatches twice. Invalided home, wounded, he commenced medical studies in 1917, graduating in 1923, and started practice in 1925 at Oberon; later, in 1930, buying the practice of Dr. Barton in Bathurst, which he held until his death. He was always keenly interested in community affairs in his district, and all local associations, having for their object the good of the community, had his support, and, as well, he was a prominent mason. In 1938 he was appointed to the Royal Commission on National Health Insurance, with Sir George Mason-Allard and Judge Dethridge. Mulvey was a distinguished general practitioner. He was one of the pioneers of FOCLA, being prominent in the organizing of the first course at Armidale. FOCLA mourns his untimely passing, and honours his memory in this, the first FOCLA Post-Graduate Oration, which it is my great privilege to deliver. In view of the fact that we have here today a number of non-medical people as our guests, I thought that it would be wise to keep the subject matter as non-clinical and non-technical as possible, and so I decided to speak on the early days of medicine and about some of the men who have made modern medicine so spectacularly efficient and have laid the foundations of its mighty structure.

Medicine and surgery have been practised from the beginning of time. It is known from the discoveries of archæologists that medicines and prescriptions were used as far back as 6000 years ago. In the tombs of the Pharaohs potions were placed in the deceased's coffin, to be part of his equipment for use during his journey into the shades. There is evidence that in 2000 B.C. there was a medical school in China, where that remarkable man, Hua Tu, was using a form of anæsthesia to assist him to perform his numerous operations. Unfortunately, owing to the teachings of Confucius, surgery did not progress in China, because of the Confucian belief that the human body was sacred and should not be mutilated. In the excavations of Pompeii a doctor's surgery was found and also evidence of a consulting room. The consulting room was also an operating theatre and was open to the public, who were critical of the efforts of the surgeon; as the French would say, they "assisted" at the performance. In the Middle Ages, A.D. 400 to 500, much progress was made in medicine and surgery. However, Galen antedated this period as one of the great names in medicine. He lived in the second century. At Salerno in Italy, in the eighth century, there was a flourishing medical school. It was the first medical school to give a degree, and trained thousands of students during its 800 years of existence. They took an oath of service to the poor without payment. The physicians of the period were the clergy, who were the only educated men of the time, and to them the poor looked for relief in their illness. They continued this work until the reign of Pope Boniface VIII, who held that they were paying too much attention to medicine and neglecting their spiritual duties. This left the field open to the barber surgeons. The University of Paris, founded by Charlemagne in the twelfth century, added the faculty of medicine to the other three faculties, of arts, divinity and law. In consequence many began to study the works of Galen and of Hippocrates. The local barber, by virtue of his dexterity with sharp instruments, began to practise on all who had the courage to suffer his ministrations. During the Renaissance period, 1400 to 1600, medicine continued to progress, as did the arts and sciences. We have during this time one of the greatest names in medicine, that of Ambroise Paré, who died in 1590, aged eighty years. He remarks in his memoirs that he was a resident doctor in the Hospital of Paris for three years, and that he had the opportunity to treat disease and dissect a limb or two. He served under four kings and was one of the first army surgeons of modern times, although he was thousands of years behind the Grecian surgeons, who served in the Battle of Troy.

In the early fifteenth century it became obvious to the surgeons and physicians that they should combine their beliefs and knowledge for their mutual protection and lay down a code of ethics. This code was laid down in 1423 and lasted for two hundred years. However, it was not until 1745 that the Royal College of Surgeons came into existence and received its Royal Charter. It was about this time that doctors, in common with other people, such as shopkeepers and innkeepers and the like, decided to have a sign. Some doctors' signs were weird and wonderful, and often a Turk's head was used with the tongue poked out, a huge pill being balanced on the tip. The Turk's head represented rhubarb, opium and other drugs. However, the first brass plate appeared in Shakespeare's time, about 1600. It is interesting to recall, too, that the barber's pole as we know it today stems from the stick used by the barber surgeons of the time, whose patients, submitted to the blood-letting operation, were required to keep squeezing the stick to make the veins stand out. The spiral stripes on the stick represent the tourniquets which were put on the upper part of the arm to assist in the bleeding process.

Medicine continued to progress in the seventeenth century. The doctor of that period usually wore a top hat, wig with bun or queue, silk hose and knee breeches with buckled shoes. His visiting coach was as ornate as his dress, and he had as many horses as his purse would allow, usually two to six. He also carried a silver-topped cane in which was a recess called a vinaigrette, which held a sponge soaked in vinegar; this he held in front of the nostrils to protect him from noxions odours and infection when visiting patients. Sir Thomas Brown is the best known medical name of this period; he is famous for his authorship of the classic "*Religio Medici*". Another great name in medicine at this time was Sir Hans Sloane, who was famous as a doctor, and who, although abstemious in his own habits, was wont to give lavish parties for prominent people in the city. At one of these the great Handel was clumsy enough to drop a buttered muffin on one of

[1] The Roy D. Mulvey Memorial Oration, delivered at the fourth annual post-graduate course of the New South Wales Federation of Country Local Associations, Wollongong, March 14 to 18, 1955.

Sir Hans Sloane's treasured medical books; for this the good doctor never forgave him. Sloane was a great physician and a student of Sydenham. John Ratcliffe and Arbuthnot of Edinburgh were also great figures of the time.

The eighteenth century is chiefly remembered for the growth of the custom of blood-letting. Doctors then, as they are today, were so wise that they thought they could correct Nature, and so the universal custom of blood-letting grew. It was very necessary, as most people of that period were gross eaters. It was the age of gluttony. Huge meals were eaten and washed down with large quantities of port wine and claret. There is no doubt that many a patient was saved from death from apoplexy by this custom of blood-letting; a man of those days would report monthly to his surgeon for this, as we would to our barber for a haircut. Oliver Goldsmith was a medical practitioner in this period; but he is better known for his literary work than for his medical acumen. It is the glory of the medical practitioner of the eighteenth century that he laid the clinical foundations upon which the nineteenth century built the brilliant superstructure. What was it that the practitioners did? They laboured as individuals, with relatively little interrelationship or coordination. They often worked in the dark, and almost always inductively. They began to use the autopsy to check their treatment; they introduced methods of precision in diagnosis; and almost unconsciously they began to apply the new physiology to their clinical work. It is only now that, looking back, we can weave their individual labours into terms of principle and collective advancement. They builded better than they knew. Turning the matter over in my mind, and as the result of both experience and reflection, I should say that quite apart from their contribution to culture and literature, to therapeutics and to surgery, they moved English medicine forward in the following six preventive directions:

1. They explored the circumstances of epidemic disease and observed the causative relation of external environment to it, including the effect of climate.

2. They introduced the principles of medical notification, of isolation, and of fumigation and disinfection, though their disinfectants were probably not germicidal.

3. They advocated an improved and enlarged dietary, combined with restriction of spirit drinking. Tea, coffee, vegetables and fruit were warmly recommended by them.

4. They initiated industrial welfare, and were the prime movers in the health and sanitary control of the factory system.

5. They began the reformation of midwifery, first by undertaking the practice of midwifery themselves, and secondly by improving the methods of delivery and the care of lying-in women.

6. They first systematically attacked the problem of infant mortality.

The nineteenth century witnessed further progress, and during the reign of George III, the sixty years from 1760 to 1820, there was much improvement in surgical and medical technique and in the refinement and manufacture of drugs. It was in this period that the great John Hunter flourished—one of the medical immortals. He laid the foundation of anatomy and pathology.

I shall now say something about surgery. Surgery has been practised from the earliest times. Celsus, 25 B.C. to A.D. 50, laid down one of the earliest definitions of a good surgeon. He said that a surgeon must have a strong, steady and intrepid hand, and a mind resolute and merciless, not giving heed to the judgement of the common people. Guy de Chauliac in 1300 laid down that a surgeon should be learned, skilled, ingenious and of good morals, bold in things that are sure, cautious in things that are dangerous. George Baker in 1540 adjured his students in these words: "Think what a precious thing you work upon. It is the Temple of God, His own image, the most precious creature that God ever made." John Bannister's pronouncement was as follows: "Great pity and truth it is that so many idle idiots and erroneous asses are permitted to practise this art of great difficulty." Finally, Lord Lister (1827 to 1912), whose work ushered in the new era in surgery, had this to say: "To intrude an unskilled hand into such a divine piece of mechanism as the human body is indeed a fearful responsibility."

Many of the leading surgeons in the late eighteenth century took in apprentices. The apprentice was taken into the doctor's house. The master was responsible for his material and spiritual welfare. He took him with him on his rounds. The apprentice carried a notebook and took down what his mentor said about the patient. His other tasks included sweeping out the office and compounding pills and potions, and it was a red-letter day when his master amputated the limb of a patient, because he was allowed to dissect it and to learn its anatomy. The barber surgeons who antedated this period were known as "leechers", probably from the common practice of applying leeches to engorged and inflamed tissues. I think most doctors these days would object to being called a "leech". The general practitioners of the time were barbers. Plastic surgery is thought to be a very modern development of the art, but it is known that it was practised extensively in the fifteenth century. One plastic surgeon, well known at the time, was Branca in Sicily, who was called by an historian of the period "a celebrated surgeon, who replaced ears, lips and noses, and who had learned the art of restoring the nose by supplying it from the arm of a patient, or by infixing upon the part the nose of a slave". King Henry VIII had his own barber surgeon. This functionary was required to be in attendance on His Majesty at all times, and to have ready the basins, potions, scissors, combs, trephines, needles, cupping paraphernalia and so forth. He had to be prepared at any time to cut the King's hair, trim his beard or perform an operation on him if required. The King also instructed him to speak moderately and to mind the company that he kept, being careful to avoid people who indulged in idle gossip and women of loose character.

Surgery reached its zenith in the time of Lister. This great doctor transformed the face of medical work, profiting by the work of Pasteur. In Edinburgh he studied under Syme, that greatest of all Scottish doctors, and he married Syme's daughter. He founded the system of aseptic surgery in March, 1865, and used carbolic acid in the case of a compound fracture. A cynic has said that science knew everything about the universe, the stars, the moon and the sun, but it did not know how to render a pin-prick harmless or to dress a finger till Lister came. Syme performed the first surgical operation under antiseptic conditions in 1877, using his famous carbolic spray. He had an immense audience of students and doctors, but not all were convinced by the new theories, and one leading surgeon was observed to turn his back on the master, put his thumb to his nose and leave the operating theatre. The leading physician of the time, who was quite ignorant of the antiseptic theory of Lister, was so excited when he saw the knee joint open, preparing for the wiring of the patella, that he tried to put his unsterilized fingers into the wound. Syme, however, knocked him down before he could get his fingers into the wound; then, recarbolizing his hands, he continued the operation, and the patient was discharged cured, the wound having healed by first intention in three weeks. Previously this operation was attended with a morbidity or mortality of over 90%. Truly a new day had dawned for surgery.

Side by side with the growth of modern surgery there were developments in hospitals. One of the most famous hospitals of early times was that of the Knights of Saint John of Jerusalem. Regulations from this hospital have been handed down to modern times. Separate sheets and covers were used on beds, and other precautions were taken to prevent infection. Another great hospital was the Hospital of the Knights of Saint John at Rhodes. Rahere is said to be the founder of St. Bartholomew's Hospital. After a pilgrimage to Rome he is said to have seen an apparition who said: "I am Bartholomew, the Apostle of Jesus Christ and know you that by command of the Trinity

I have chosen a place in the suburb of London at Smithfield where in my name thou shalt found a Church and it shall be a House of God." The *Hôtel Dieu* in Paris was founded also around about this time, close to the river Seine. It was staffed by nuns, who did all the work at the hospital and the laundry of the patients. It was hopelessly overcrowded, having four or six patients in each bed regardless of disease, the living with the dying.

Paralleling all these developments in medicine and surgery was the development of anæsthesia throughout the centuries. Whilst it is common to believe that the science of anæsthesia is of comparatively recent development, nothing could be further from the truth. It is known from the annals of the Han dynasty that anæsthesia was practised extensively by the great Chinese doctor, Hua Tu, over two thousand years before the birth of Christ. This great man mixed an infusion of certain herbs with wine and gave the potion to his patient, who in a short time was sleeping soundly, completely insensible. Hua Tu performed numerous operations and is known to have opened every cavity in the human body. Other doctors in the early ages used forms of anæsthesia, mostly a mixture of drugs with alcohol; but it was not until the early fourteenth century that inhalation anæsthesia was first practised. The method used was to infuse certain herbs in water or wine, dip a sponge in the mixture and allow the fluid to evaporate. When an anæsthetic was required, the sponge was plunged into boiling water and the fumes which were given off were held close to the patient's nose; after a short time he was said to be sound asleep and ready for the operation. However, it was not till 1846 that the modern inhalation anæsthesia became certain in its results and safe in its application, mainly owing to the work of two great men. The first was William T. Morton, a surgeon of Boston, United States of America. He, whilst experimenting with different volatile liquids, put his handkerchief dipped in the liquid over his face, and in a short time after inhaling the fumes he became unconscious. Ether had been discovered. Morton was wildly excited at this experiment, and could hardly wait for a patient to show up so that he could try the anæsthetic. Very soon afterwards a man who had a roaring toothache turned up and asked Morton to mesmerize him and extract the offending tooth. Morton said, "I have something better here than mesmerism", and anæsthetized him with ether. The offending bicuspid was painlessly removed. The first major operation under ether anæsthesia was performed on October 17, 1846, in America, and the first in Europe on December 21, 1846. It was performed by the great surgeon, Liston, who announced to the crowded theatre: "Gentlemen, we are about to use a Yankee dodge for making men insensible." The operation, an amputation of the thigh, was painlessly performed, and the patient woke up and asked when the operation was going to be performed. It was hard to convince him that the operation was over. Liston was delighted with the anæsthesia and remarked: "Gentlemen, there must be something in this Yankee dodge. It is better than mesmerism." Chloroform came closely on the heels of ether, and in 1847 Sir James Young Simpson first used chloroform in an obstetric case. There were plenty of critics for his temerity in interfering with the designs of the Lord, who ordained that woman must in labour and anguish bring forth children. However, Simpson was equal to his critics and referred them to the twenty-first verse of the second chapter of the Book of Genesis, recounting how the Lord, after putting Adam into a deep sleep, removed a rib from his side. Simpson was winning his battle now, and his victory was complete when Queen Victoria allowed herself to be anæsthetized during the birth of a child. However, modern anæsthesia has outdistanced ether and chloroform, great and wonderful as these drugs are. It is now no exaggeration to say that practically any operation can be performed if it depends only on the safety of the anæsthetic. Anæsthesia has really outdistanced surgery, and we have reached a stage at which, as a famous surgeon remarked: "We have made surgery safe for the patient; now we must make the patient safe for surgery."

RECENT ADVANCES IN ARTHRITIS.[1]

By L. J. A. PARR,
Sydney.

Some Ætiological Considerations.

WHEN the researches of Phillip Hench and his colleagues directed the spotlight on the antiphlogistic properties of cortisone and ACTH, it was thought by some rheumatologists that rheumatoid arthritis in some way or other was bound up with metabolism of the steroids. However, there was never any claim by Hench that steroid therapy was curative; he stressed the fact that, like the protection against fire afforded by an asbestos suit, steroids gave protection against the fires of the disease but still left the latter smouldering. It is well for us to recall to memory that the following factors are pro-remissive of rheumatoid arthritis: typhoid fever, pneumonia, pyelitis with fever, hypoglycæmia and coma, pregnancy, operative procedure and injury, jaundice and starvation. Other conditions may be anti-remissive and either provoke or produce an exacerbation; these are sudden mental shock, thyrotoxicosis, malignant disease, overdosage of streptococcal vaccine, acute tonsillitis, sinus infection, removal of septic teeth without a sulphonamide "cover", acute cystitis and uterine infection following abortion. Over the last twenty-eight years of observation of rheumatoid disease I myself have seen all these either precede or produce an exacerbation of rheumatoid arthritis. During this same period I have never observed any exacerbation of this disease caused by boils or severe carbuncles, the hæmolytic *Staphylococcus aureus* not being a factor in causation; but infections by the *Streptococcus hæmolyticus* or *viridans*, the latter common in the periapical infections of teeth, are a common cause of exacerbations. However, the failure by numerous investigators to obtain positive cultures from affected joints (although in three of my cases I have grown a pure culture of *S. viridans*) suggests that if infection is a factor, the ætiological agent is either an unidentifiable organism or virus or possibly an antigen-antibody reaction. No one can deny that in a small percentage of cases dramatic recovery follows the removal of foci of infection, whilst all rheumatologists can testify to repeated failure of such therapy. The high titre of antistreptolysin and antifibrinolysin in the blood of patients suffering from rheumatoid arthritis, although lower than that in the blood of rheumatic fever subjects, only adds confusion to the ætiology, especially as the *S. hæmolyticus* is an uncommon organism in any demonstrable foci. The sudden onset with moderately severe febrile reaction in some cases of rheumatoid arthritis following localized infection or exposure to chill would seem to point to a bacterial or virus factor in the disease.

In a recent examination of 150 cases, I have found that in the largest percentage the condition began in a large joint, and that within a space of a few years in 75% it had developed into the typical bilateral symmetrical rheumatoid arthritis with small-joint involvement. My experience in general practice a number of years ago led me to believe that foci of infection should be removed very early in the disease, and that remission or cure was often obtained as a result, but that once the pattern was that of bilateral symmetrical small-joint involvement the results of removal of septic foci were generally unsatisfactory. I have surmised that there had occurred, possibly in the enzyme systems, some changes which perpetuated the disease and militated against recovery. However, I have examined patients who have had complete unaccountable remissions of severe deforming rheumatoid arthritis of many years' duration, which shows that even in the advanced cases with generalized deformity, the fires of the disease can disappear without any possible assignable reason. I have also observed a sudden recurrence of the disease after years of remission, which in some cases was caused by upper respiratory infection, whilst in others no

[1] Read at the fourth annual post-graduate course of the New South Wales Federation of Country Local Associations, Wollongong, March 14 to 18, 1955.

cause was discoverable. The conception of rheumatoid arthritis as one of the collagen diseases does not help us any further along the ætiological road, and the fact that in this disease as in other diseases large or small aggregations of lymphocytes are in muscles, around tendons, blood vessels, sympathetic ganglia, and nerves and in synovial membrane has not clarified the ætiology. Is there any evidence of glandular disease in rheumatoid arthritis?

There are some pertinent considerations which do not suggest a clearly defined role for the surprarenal gland in rheumatoid arthritis. Rheumatoid arthritis is rare in Addison's disease, in which the glucosteroids may almost disappear from the urine. The suprarenal gland in rheumatoid arthritis, even after prolonged disease, is histologically normal, and further responds normally in about 85% of cases to the subcutaneous injection of adrenaline. Hypoglycæmia is common in Addison's disease but rare in rheumatoid arthritis, whilst in the former muscle flaccidity and hypotonicity are present, whereas in the latter there is often muscle spasticity. Further, in many cases of rheumatoid arthritis it requires an amount of cortisone sufficient to produce Cushing's syndrome to relieve the disease—in other words, a mildly pathological dose. It has recently been shown that in a number of juvenile rheumatoid arthritics the steroid level is consistently low, as it is in rheumatic fever. Norymberski, Stubbs and West (1953) have published the result of estimation of the ketogenic steroids in the urine, which, they state, is indicative of hydrocortisone excretion. In rheumatoid arthritis and ankylosing spondylitis they found that the output was about 40% lower than normal. According to West, treatment by ACTH causes an increased urinary output. Confirmation of these findings may be of great ætiological importance.

Selye has shown recently that jaundice will inhibit inflammation chemically induced in the rat. Jaundice produces increased ACTH secretion; but the main effect, according to Selye, is primarily a sensitization of the tissues to the antiphlogistic corticoids—the so-called "A-CC effect" (antiphlogistic-corticoid-conditioning factor). Temporary remission of rheumatoid arthritis brought about by jaundice is a time-honoured observation.

The work of Ungar on the endocrine function of the spleen, published in 1945, appears to have passed almost unnoticed in medical literature. He showed that the spleen under stimulation of the pituitary-adrenal axis produced a substance which reduced capillary permeability and inhibited histamine release by blood cells. The substance was called splenin A, and it was found that another substance, splenin B, also obtained from the spleen, had opposing qualities.

In starvation experiments on guinea-pigs, it was found that there was a reduction of bleeding time, accompanied by the formation of splenin A, beginning at the fourth day and reaching its maximum on the sixth. Starvation for four to six days was an old anti-rheumatoid therapeutic procedure of limited usefulness. Both Ungar and Greene have shown that in rheumatoid arthritis there is an absence of splenin A in the serum and a considerable increase in splenin B. In malignant disease there is a considerable increase in splenin B and an absence of splenin A, and it is significant that malignant disease aggravates rheumatoid disease. In thyrotoxicosis there is a considerable increase in splenin B and a reduction in splenin A, and, as was previously stated, thyrotoxicosis aggravates rheumatoid arthritis. The potentiation of this disease by thyrotoxicosis may be due to the additive effect of suppression of the secretion of splenin A. Splenin A deserves a therapeutic trial in rheumatoid arthritis.

Some recent research on hyaluronic acid and hyaluronidase may soon clarify some of the disturbances in physiology in the rheumatic diseases. It has been observed that there is a rise in the serum inhibitors of hyaluronidase in acute and chronic rheumatic diseases, and it has been shown that there is an increase in the mucopolysaccharides in the inflammatory synovial fluid, but that the degradation of these substances is delayed by the rise in hyaluronidase inhibitors. This may be of more importance in osteoarthritis than in rheumatoid arthritis. The recent report of the development of a syndrome resembling typical rheumatoid arthritis and in some cases disseminated *lupus erythematosus* after treatment for hypertension with hydralazine, of its remission on cessation of the drug and of its recurrence on recommencement of the treatment, gives to the experimentalist methods for the production of the disease and for study of the enzyme systems involved. In summary it can be stated that the horizons of the causation of rheumatoid arthritis are widening, and that future research should soon produce knowledge of important enzyme systems involved in its pathology. The lack of clarity concerning causation suggests that the object of treatment should not be to throw overboard methods proved by experience to be beneficial, but rather to add to our therapeutic armamentarium.

The consideration of some basic facts in the knowledge of the disease as well as of the newer therapeutic discoveries of the last five years compels a cautious approach to the handling of these cases of rheumatoid arthritis, which are so divergent in their objective clinical manifestations.

The therapeutic armamentarium of the rheumatologist is very much improved today in comparison with former years, as is evidenced by the following drugs and measures we employ in our private and hospital practice for the treatment of rheumatoid arthritis. (i) Sulphonamides: benzyl sulphanilamide ("Proseptasine"—in our experience the most successful); sulphadiazine and sulphadimidine and rarely sulphathiazole (dosage, 1·5 grammes *per diem*). (ii) Sulphones: "Promacetin" (acetosulphone) or "Sulphetrone" (1·5 grammes *per diem*); (iii) Salazopyrin (salicyl-azo sulphapyridine—three grammes daily), useful in arthritis associated with ulcerative colitis; (iv) Mepacrine (0·1 gramme twice daily), chloroquine (0·2 gramme twice daily); (v) insulin, used to increase appetite and weight; (vi) adrenaline (1:1000, preferably in oil), five to seven minims twice or three times a day; (vii) calcium gluconate (10 cubic centimetres of a 10% solution once a week); (viii) gold (still a valuable remedy in selected cases); (ix) sodium salicylate or aspirin (40 to 60 grains daily as required); (x) thyroid and ovarian hormones when indicated; (xi) "Butazolidin", a drug of great usefulness; (xii) blood transfusions, useful in severe anæmia; organic iron, of limited use; (xiii) hormones—cortisone, hydrocortisone and ACTH; (xiv) liver, especially crude liver such as "Campolon" or similar preparation; (xv) physiotherapy; (xvi) rest and orthopædic measures.

Acute Rheumatoid Arthritis.

Acute rheumatoid arthritis calls for special comment. In its acute onset, fever and high erythrocyte sedimentation rate it resembles rheumatic fever; but the absence of a hæmolytic streptococcus in the throat and of cardiac signs, the failure to respond to heavy salicylate dosage, the fixity of joint involvement and the type of joint involved clarify the diagnosis. Admission of the patient to hospital, complete rest and, if necessary, splinting of the joints are important. Sulphonamides (two grammes three times a day) and salicylates in full dosage are employed, besides adrenaline (0·5 cubic centimetre in oil three times a day). The stimulation of the pituitary-adrenal axis by adrenaline is necessary in such cases. In successful cases the temperature will begin to fall in forty-eight to seventy-two hours after the commencement of administration of adrenaline. Ascorbic acid (one gramme per day) is given, and blood transfusions can be employed when the fever subsides if necessary. They are of great value in speeding recovery in such cases. Convalescence is aided by a protein-rich diet, whilst "Methionine" (four grammes per day) and insulin to improve the appetite and restore lost weight may help considerably. Adrenaline is given for two or three weeks twice a day after the temperature has become normal. Hydrocortisone is employed locally for those joints in which the disease is smouldering and which are slow to recover. Sulphone therapy is tried if the sulphonamide drugs are unsuccessful. Early ambulation and physiotherapy are valuable after the fever subsides. However, hormone therapy (cortisone or ACTH or both)

is employed only when the above-mentioned procedures are ineffectual. Reduction of the dosage of sulphonamide drugs to 0·5 gramme three times a day is effected after the temperature is normal, and this amount is employed until the erythrocyte sedimentation rate returns to normal and clinical evidence of the disease has disappeared.

Chronic Rheumatoid Arthritis.

Until the ætiology is clarified, the emphasis in the treatment of chronic rheumatoid arthritis must be on suppression and control, as cure is an uncommon result.

I believe that streptococcal infection in some way or other plays a part in this disease, and long experience has convinced me that the suppression of streptococcal activity and intercurrent infection in rheumatoid disease is valuable. For many years in these chronic cases I have employed the sulphonamide drugs in doses of 0·5 gramme three times a day for long periods, because I postulated that rheumatoid arthritis was often due to an antigen-antibody reaction. Clinical improvement as well as a pronounced reduction in the erythrocyte sedimentation rate lends support to this concept.

The most successful drug has been benzyl sulphanilamide ("Proseptasine")—why, I do not know; but sulphadimidine and sulphadiazine are also of value. Supplementary therapy consists of the administration of yeast or liver, both of which help to prevent blood dyscrasias. I have administered these drugs to patients for years without untoward results; but rashes are a common complication and may force suspension of therapy. Improvement cannot be looked for under three or four weeks, and the patient is warned of this fact. Full blood counts and estimation of the erythrocyte sedimentation rate are essential before therapy is begun and at varying intervals during treatment. Pyridoxin, 20 to 30 milligrammes daily, can be employed if the leucocyte count tends to be suppressed, and "Campolon" is valuable if the anæmia of the disease is not relieved and the tongue is dry. "Promacetin" or "Sulphetrone" (sulphones) in similar dosage is given if the sulphonamide drugs are a failure, and I have seen some remarkable results from their use in cases of long duration in which all other remedies, including gold, had failed. Similar precautions as with sulphonamide therapy are employed. I have observed the complete disappearance of subcutaneous nodules after sulphonamide and sulphone therapy.

For patients who have lost much weight, insulin (12 units *mane* and five units *nocte*) and glucose are valuable, and the intravenous administration of 10 cubic centimetres of calcium gluconate (10% solution) once a week helps to overcome the calcium loss indicated by generalized decalcification, and to diminish muscle spasm. I find gold still of value in the periarticular variety of the disease associated with a slightly raised erythrocyte sedimentation rate. Radioactive gold, at present employed in malignant disease, may be of great value in rheumatoid disease, especially as radioactive waters and radioactive mud have been of great benefit in European spas. A clinical trial of radioactive gold in rheumatic diseases should be an immediate objective.

Salicylates, which are now known to suppress the antigen-antibody reaction, can be employed in adequate dosage—60 to 80 grains daily. Salazopyrin, in a dosage of three grammes per day, is the best drug in cases of arthritis associated with ulcerative colitis. Mepacrine and chloroquine, valuable agents in the treatment of disseminated *lupus erythematosus*, have proved to be of outstanding benefit in a number of severe cases of rheumatoid arthritis—the former in doses 0·1 gramme twice daily and the latter in twice that dosage. In some of our cases they have controlled the condition for years when all other remedies had failed—the steroids, however, not having been employed in any such case. The disease will return within four to five months after cessation of the drug. Dermatological reactions do occur in some cases, and compel cessation of treatment. After disappearance of the rash the drug can be reemployed.

"Butazolidin", closely allied to amidopyrine, has proved of outstanding benefit in the treatment of rheumatoid arthritis as well as other rheumatic diseases. Its precise mode of action is unknown, and unlike cortisone it rarely lowers the erythrocyte sedimentation rate. However, the drug is antipyretic, analgesic, anti-inflammatory and antihistaminic. It does not alter the histological condition of the joint and is of benefit whilst it is being given, but recurrence follows suspension of treatment. The toxic effects vary according to dosage, and reports of their occurrence by different authorities vary from 1·8% to as high as 52%. They consist of aplastic anæmia, agranulocytosis, gastro-intestinal hæmorrhage, toxic rash, œdema, fever, and soreness and ulceration of the mouth. From initial practice in which dosage was large (between 600 and 1000 milligrammes per day), the present procedure tends to be as follows: 600 milligrammes given daily for three weeks, and then reduction to a dose as low as is compatible with clinical improvement and minimum toxic reactions, or 200 milligrammes given initially followed by a gradual increase by 100 milligrammes a day until therapeutic response is satisfactory. However, toxicity rises rapidly with doses over 400 milligrammes a day. The blood levels are not proportionate to the intake, so that unless these are tested, the clinical response must determine the dosage. Pemberton (1954) reported that 25% of his patients had to stop therapy because of toxic reactions. In our practice we begin with 100 milligrammes three times a day and increase according to clinical response. In a number of cases of rheumatoid arthritis we have noted that liver given intramuscularly and calcium given intravenously have greatly enhanced the clinical response. "Butazolidin" can be used in conjunction with salicylate and cortisone therapy. In one case of severe rheumatoid arthritis in our practice, after cortisone had produced only mild relief and the onset of an anxiety neurosis, "Butazolidin" (400 milligrammes daily) and hydrocortisone (given by intraarticular injection), with liver and calcium produced dramatic improvement, followed by a considerable reduction of the erythrocyte sedimentation rate. We also employ "Butazolidin" with sulphonamide therapy, and have observed objective improvement with a reduction in the erythrocyte sedimentation rate. It would appear that supplementary therapy is advantageous and reduces the need for the higher toxic dosage. Constant medical supervision of the patients is necessary, and repeated blood counts are essential. Untoward symptoms and signs and especially progressive anæmia and a falling leucocyte count counsel reduction of dosage, and, if necessary, withdrawal of the drug. The drug is contraindicated if there is a history of gastric or duodenal ulceration, and in the presence of a low leucocyte count. Œdema calls for reduction of sodium chloride intake, and at times "Diamox" (250 milligrammes per day) is of decided advantage by eliminating sodium and retaining potassium.

Cortisone and ACTH Therapy.

It is apparent that the adrenal corticoids, cortisone or hydrocortisone and ACTH, are only palliative or suppressive and not curative. Continuous therapy in doses sufficient to cause the desired relief is associated with undesirable effects from the drugs in over one-third of the cases. An interim report of the American Rheumatism Association's Committee gave only 23% of patients as stopping therapy because of undesirable effects; but the report also revealed that 46% of patients after prolonged therapy showed one or more undesirable effects considered of major significance.

The complications of prolonged high cortisone dosage are as follows: (i) osteoporosis; (ii) psychotic episodes—insomnia, restlessness, *et cetera*; (iii) gastric or duodenal hæmorrhage; (iv) elevation of the blood pressure; (v) œdema with weight gain—moon facies; (vi) hyperglycæmia and glycosuria; (vii) spontaneous fractures, especially in the aged; (viii) hypertrichosis and acne; (ix) fulminating infection with masking of the serious nature of this complication (perforation and peritonitis following acute appendicitis); (x) tuberculosis—activation of a quiescent lesion; (xi) buffalo hump—fat deposition at the back of

the neck; (xii) suprarenal collapse following surgical procedure.

It is wise to examine the patient's chest radiologically before cortisone or ACTH therapy is begun, and to inquire whether there is any history of tuberculosis affecting the patient and the family.

It has been our practice to use cortisone only when we find that no other therapeutic procedure already outlined is efficacious. All rheumatologists are aware that in the vast majority of cases the disease goes on slowly underneath the suppressive steroid therapy. The indication for this therapy is the failure to control the disease by other measures after adequate trial. However, there are conditions such as periarthritis of the shoulder, calcific tendinitis and the shoulder-hand syndrome which will respond and remit under cortisone therapy more quickly than under other measures, and therefore such therapy can be used initially unless there are contraindications for such short-term treatment.

The long-term treatment is the most important from the point of view of complications, and also because it concerns the patient whose disease is proceeding rapidly in spite of all other therapy carefully and persistently tried. It should not be undertaken lightly, because once begun it can rarely be stopped without the disease galloping ahead. Recommended therapeutic dosages are still variable. Most rheumatologists prefer 100 to 200 milligrammes per day, with prompt decrease as soon as possible to 50 or 75 milligrammes per day. Certain potentiating drugs have been suggested such as aspirin, paraamino benzoic acid, adrenaline, gold and "Butazolidin"; of these aspirin, in doses of 60 to 80 grains a day, is preferred by most. Some authorities, including Kinsell, give ACTH and cortisone intravenously. The dosage recommended by Kinsell is 600 to 800 milligrammes daily, the object being to reduce the erythrocyte sedimentation rate to normal. Kinsell employs a diet of low sodium and high protein content, and gives potassium chloride by mouth. To achieve maintenance a dose of 50 to 75 milligrammes daily as quickly as possible is his aim.

Most authorities prefer the smaller doses for maintenance therapy, seeking to suppress the disease and avoid side-effects. The sooner a maintenance dose of 50 milligrammes per day or less is reached with symptomatic control, the better for the patient. As cortisone suppresses the suprarenal cortex, it is advisable to use ACTH, preferably the long-acting variety, giving a short course from time to time. Approximately seven days of ACTH therapy are required before the cortex responds normally. We use 20 to 40 milligrammes of "Androcort" or "Corticotropin Z" per day. Another method is to give hydrocortisone intraarticularly for those joints in which the disease is still active in spite of a maintenance oral dose of cortisone. We have done this on many occasions with success and with control of residual inflammation. Such a procedure helps to diminish the number and severity of complications due to cortisone, and permits a heavy concentration of the hormone in the proximity of the damaged tissue, the seat of resistant disease.

Hydrocortisone given orally has been stated by many to be superior to cortisone and more potent when used in the same dosage. This would seem to be confirmed in two cases in my practice in which it has been tried recently. The deleterious effects of these hormones are well known. One of them, œdema, we have recently observed, can be easily controlled by "Diamox", 250 milligrammes every second day; this inhibits the carbonic anhydrase activity in the kidney. However, prevention of osteoporosis induced by cortisone and ACTH is extremely difficult, and, although there are opposing opinions, I think that in the treatment of older people methyl androstenandiol, 20 milligrammes daily, and one gramme of calcium gluconate are helpful. Infection of a massive kind can occur during cortisone and ACTH therapy, and requires adequate steroid therapy to prevent suprarenal collapse and massive doses of antibiotics—at least double the usual dosage. Continual vigilance is necessary, not only because of the masking effect of the drugs, but because they seem to potentiate infection. In those patients who have had prolonged cortisone therapy and for whom surgical intervention is necessary, the Mayo Clinic's procedure is as follows: 200 milligrammes of cortisone are given intramuscularly forty-eight, twelve and one or two hours before operation, and cortisone therapy is continued for three or four days after operation on a reduced dosage. ACTH can also be employed with advantage.

I always inject hydrocortisone, in a dose varying from 0·25 to 2·0 cubic centimetres as required intraarticularly after anæsthetization of the skin and deeper tissues with procaine. In the case of the knee, to make certain that the joint is entered—that is, if no synovial fluid emerges—I inject 10 cubic centimetres of air and watch for the ballooning of the joint. The hydrocortisone should flow in easily. I have injected hips, knees, elbows, shoulders, wrists, and metacarpophalangeal and interphalangeal joints. Weekly doses are required. On rare occasions inflammatory reaction may follow. With ordinary care the procedure is simple and painless and produces great relief. It is of very little value in osteoarthritis, but is of value in Dupuytren's contraction.

Physiotherapy, mainly the application of heat and histamine ionization, although less employed than formerly, is still of value, and muscular reeducative exercises are helpful. Newer steroids are on the way, but at present are only in the trial stage. They are for oral use as well as for intraarticular injections. Some are very promising, having increased anti-rheumatoid effect and reduction of complications.

Ankylosing Spondylitis.

Ankylosing spondylitis is mostly regarded by the American rheumatologists as a spinal variant of rheumatoid arthritis; but this conception is losing ground to the other belief that it is a separate entity. The peripheral joint involvement of ankylosing spondylitis is now more widely recognized, and the frequency of iritis and iridocyclitis (about 10%) is more frequently commented upon in the literature. With regard to treatment, the pendulum has swung from emphasis on rest with resultant fusion to that of a greater activity with consequent improved results. The importance of back supports, exercise and postural drill is still being stressed, whilst manipulation has now no part in therapy. "Butazolidin" has proved to be of outstanding benefit, especially when the hips, knees and feet are involved. I have found it more valuable than cortisone in many cases. For some patients not responsive to deep X-ray therapy, cortisone or ACTH therapy is valuable; but "Butazolidin", preferably with calcium gluconate given intravenously, should be first employed. Iritis and iridocyclitis call for local and oral cortisone or hydrocortisone therapy, and no time should be lost in employing the latter. In one case in our practice in which the knee joints had been persistently distended for over five years and had defied all therapy, including X-ray treatment, "Butazolidin" therapy in two months produced almost normal knee joints, which have remained so for two years whilst a daily maintenance dose of 200 milligrammes has been employed.

Treatment of Osteoarthritis.

It would seem as if the era of therapeutic passivity with regard to osteoarthritis is on the way out. Owing to lack of time, I cannot deal with spinal osteoarthritis and all its implications and therapy. There are two important medical gains in the treatment of osteoarthritis over the last few years. "Butazolidin" has emerged as a drug which can have a profound effect by reducing pain and stiffness in osteoarthritis and increasing movement. The dosage is similar to that for rheumatoid arthritis. I have found that many patients, victims of osteoarthritis of the hips and knees, can be maintained in relative comfort for long periods on a dose of 200 milligrammes of "Butazolidin" per day. It is of very great value in secondary osteoarthritis of the hips and knees following rheumatoid arthritis. Orthopædic measures and physiotherapy are still employed, but medical therapy has been greatly enhanced. The usual hæmatological precautions

are necessary. Today "Butazolidin" is the drug of choice in osteoarthritis. Another remedy that may prove of value in the near future is hyaluronidase, which can be injected into the joint as long as procaine is added to reduce any painful reaction. After one or two such injections of 10 units the enzyme is injected subcutaneously over the joint, procaine still being mixed in the same syringe. The injections are given twice a week, then once a week, and for as long as they are required. This treatment has been suggested as a result of the discovery that hyaluronidase is deficient in chronic arthritis. Aspirin and pethidine are often helpful. Cup arthroplasty and the use of the acrylic head are notable advances in the surgical treatment of hip joint disease.

Senile Osteoporosis of the Spine.

Senile osteoporosis of the spine, important in an aging population, calls for a number of measures. Spinal support is required to relieve pressure and reduce muscle spasm. Secondly, some measures must be taken to retain calcium. Most of these patients are past both the menopause and the adrenopause, with the loss of the nitrogen and calcium retaining androgen steroids. The hirsutism so commonly experienced when methyl testosterone is given is seen only rarely with methyl androstenandiol, which is more potent than the former with regard to calcium retention. When this is given by mouth in doses of 50 milligrammes per day for a period of three or four weeks, and later reduced to a daily maintenance dose of 15 to 30 milligrammes, a patient can be maintained in relative comfort. If there are crush fractures and severe pain is experienced, bed rest and a slow reduction of spinal flexion will be necessary, so that admission of the patient to hospital is imperative. With regard to diagnosis, many of these patients are falsely labelled as suffering from spinal osteoarthritis; but the pain and stiffness are relieved by rest and accentuated by movement, features inconsistent with rheumatic causation. Morning stiffness which disappears as activity is assumed is diagnostic of active rheumatic disease, and one must be careful to take notice of the symptomatic history as well as of the radiological appearances. In elderly patients who are suffering from both diseases, the history of early morning stiffness and pain which disappears after an hour or two and recurs later in the day, often necessitating bed rest in the afternoon, is diagnostic of active osteoarthritis associated with osteoporosis.

Reports of Cases.

Sulphonamide and Sulphone Therapy.

A female patient, aged twenty-three years, had suffered from generalized rheumatoid arthritis of the upper and lower limbs for two years. She had lost over 14 pounds in weight. On examination on August 1, 1947, she was found to be suffering from typical rheumatoid arthritis of the fingers, wrists, elbows, shoulders, knees, ankles and toes. There was considerable diminution of all movements, and her hands were cold and clammy. The erythrocyte sedimentation rate was 35 millimetres (Westergren) at one hour.

She was given "Proseptasine", 0·5 gramme three times a day, and yeast. Three months later the erythrocyte sedimentation rate was 20 millimetres in one hour. She made considerable clinical improvement for the next three years under sulphonamide therapy; the erythrocyte sedimentation rate was five millimetres in one hour on May 19, 1950, and she had gained five pounds in weight. Chrysotherapy was added in an attempt to clear up some residual stiffness, and insulin to improve her weight. Two years later, on May 20, 1952, her erythrocyte sedimentation rate was seven millimetres in one hour. Gold and insulin therapy was suspended after seven months.

On March 22, 1953, her weight gain was 13 pounds. The condition in her joints flared up slightly again even whilst she was taking "Proseptasine". A change was made to "Promacetin", 0·5 gramme three times a day. Three months later the patient reported that she was well. On March 12, 1955, the erythrocyte sedimentation rate was six millimetres in one hour, and the patient had gained 17 pounds in weight; she had no pain and no stiffness, required no analgesics, and carried on all household activities without any trouble.

The total period of sulphonamide and sulphone therapy is now over seven and a half years, and the patient is clinically well, with only a residuum of deformity in one wrist. She has no pain and no morning stiffness. There have been no complications.

Mepacrine Therapy.

A male patient, aged fifty-six years, had suffered from rheumatoid arthritis for four months before consulting me on September 8, 1952. The disease came on suddenly, and attacked both hands and wrists, and the left ankle and right shoulder. The erythrocyte count was 3,800,000 per cubic millimetre, the leucocytes numbered 11,600 per cubic millimetre, and the erythrocyte sedimentation rate was 30 millimetres in one hour (Westergren). He was given "Promacetin" without any relief, and mepacrine, 0·1 gramme twice daily, was ordered on October 10, 1952. On April 24, 1953, the erythrocyte count was 4,300,000 per cubic millimetre, the leucocytes numbered 6800 per cubic millimetre, and the erythrocyte sedimentation rate was six millimetres in one hour. The clinical result was excellent; all stiffness, pain and swelling had disappeared except some thickening and reduced movement of the left wrist. Mepacrine therapy was suspended on June 21, 1953, because of dermatitis due to the drug. Five months later the disease began to recur. On January 21, 1954, the erythrocyte sedimentation rate had risen to 14 millimetres in one hour, and mepacrine therapy was reinstituted. Four months later the joint pain and swellings were well controlled. On February 28, 1955, the erythrocyte sedimentation rate was 16 millimetres in one hour; but his clinical condition was excellent, except for some slight stiffness and swelling of the left wrist. There has been no recurrence of the rash. He is now able to carry on his ordinary occupation as an orchardist.

This case illustrates clinical control without complete control of the pathology of the disease.

Sulphone Therapy.

A male patient, aged forty-nine years, had suffered from generalized rheumatoid arthritis of the upper and lower limbs for nine years before consulting me on February 7, 1950. He had been given chrysotherapy on and off for years, having had four courses of gold. All the joints of the upper and lower limbs were affected except the hip joints, and the appearance of his hands was suggestive of *arthritis mutilans*. There were extensive nodules on the elbows and hands. The pain necessitated his taking 30 to 40 grains of aspirin daily. The erythrocyte sedimentation rate was 24 millimetres in one hour (Westergren). He was given "Proseptasine", yeast and vitamin *C*, and he noted an improvement after one month. Gold and calcium gluconate (10% solution), the latter given intravenously, were added on September 4, 1950, after the erythrocyte sedimentation rate had fallen to seven millimetres in one hour. It was five millimetres in one hour on February 16, 1951. An attack of virus hepatitis produced a severe flare-up of his joint condition in May, 1952, and cortisone (75 milligrammes per day) was prescribed by his local doctor.

On June 21, 1952, "Promacetin" therapy in a dosage of 0·5 gramme three times a day was commenced, in conjunction with 75 milligrammes of cortisone daily; the latter was reduced to 12·5 milligrammes twice a day on August 24 (approximately two months later), and it was entirely suspended on October 11, the patient still continuing with "Promacetin". The erythrocyte sedimentation rate was now four millimetres in one hour, and on February 7, 1955, over two years later, it was still the same. During this period the patient was maintained on 1·5 grammes of "Promacetin" daily. He has gained 21 pounds in weight and can work as a motor mechanic all day. He has no pain or stiffness, only the residual deformity of the disease. The nodules on the right and left olecranon have entirely disappeared. He has suffered from no complications. He is of the opinion that "Promacetin" has given him more relief than any other remedy.

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THE USE OF ANTITHYROID DRUGS.[1]

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IN 1944 the first real promise of adequate control of thyrotoxicosis was given when Astwood introduced thiourea. Since then many other drugs have been introduced; but the two which have stood the test of time are methyl thiouracil and propyl thiouracil. In 1951 English makers introduced a drug now known as "Neo-Mercazole", and its clinical trials in 1952 along with its increasing use over three years have proved its superiority over the old favourites. It does not affect the white corpuscles or cause undue gland hyperplasia or vascularity, and side effects are conspicuous by their absence. The only thing holding back its general use in Australia is the cost, as it is not yet available under the National Health Service pharmaceutical benefits.[2]

Diagnosis of Thyrotoxicosis.

The ætiology of thyrotoxicosis is still wrapped in obscurity. It is generally thought that it is a "stress" disease, and certainly in many patients emotional factors do play a big part; but this cannot apply in young children (the earliest case I have seen was in a girl aged two and a half years). There is often a good deal of uncertainty about the diagnosis, especially when considerable autonomic imbalance, neurosis and some slight degree of enlargement of the thyroid are present. In recent years approximately two-thirds of the patients referred to me for an opinion as to treatment have only some form of neurosis; yet many have been treated with antithyroid drugs for long periods of time without benefit. Many have had some colloid enlargement of the thyroid since puberty. The housing problem seems to be the outstanding factor in their psychological background; many with families are living under distressing conditions and often with incompatible "in-laws". Domestic unhappiness is often a factor. A sympathetic approach by the doctor will in most cases bring about a revelation of all such factors, and before any examination is made they should be inquired into. Never rush the patient or make a "spot" diagnosis. Inquire about any variation in weight and take the weight on your own scales. After putting the patient at ease, take the pulse rate; in doing so one can often gauge whether tremor is present and what type it is. Next ask the patient to hold out one hand with elbow rigid, fingers opened out and the whole limb tensed. A fine fibrillary tremor is unmistakable, just as is the coarse intention tremor. Note the patient's movements, as restlessness and sudden jerky actions should be obvious. Try the knee jerks and look for œdema of the ankles, check the heart condition, and note the facies and especially the eyes. If they are starey or full, inquire whether this is familial or of recent development. Note the neck, inquire about any swelling and, last of all, gently examine the thyroid. I find this is the best method of approach, as it minimizes the importance of the goitre to the patient.

The following are the most important points in establishing a diagnosis: (i) the psychological background; (ii) the presence of a fine fibrillary tremor which persists with the wrist dorsiflexed; (iii) weight loss from the onset of the first symptoms; (iv) a firm hyperplastic gland with pulsation and bruit; (v) lid retraction with prominent eyes or exophthalmos. If one is in doubt and one has access to a reliable biochemist, the patient should be sent for an estimation of the basal metabolic rate, preferably after two or three days of adequate sedation. Many doctors and hospitals have acquired metabolimeters; but unless they are in charge of a trained person who understands what is meant by a "basal state", the results obtained can be most misleading and even fantastic. Use your clinical acumen, and unless the reported result coincides with your clinical evaluation it should be discarded for what is really a more reliable although somewhat slower aid to diagnosis. I refer to a four to six weeks' trial on adequate dosage of an antithyroid drug. If in six weeks at most the patient has not gained five to seven pounds in weight, if the pulse rate has not dropped to near normal, if the tremor has not abated and if a general feeling of well-being has not been established, one can be sure that there is no thyrotoxicosis, and further treatment by antithyroid drugs should be abandoned.

Primary and Secondary Thyrotoxicosis.

In the young person presenting the typical picture of Graves's disease with acute onset and rapid development of classical signs and symptoms, the diagnosis is undoubted, and this affords the best example of primary thyrotoxicosis. However, by no means all patients present with this florid condition, and especially in the thirty to forty years age period the development may be relatively slow or even insidious. The gland usually has the characteristic firm, even, general enlargement typical of hyperplasia; but if the patient has been given iodine, especially in doses over five drops (Lugol's solution) per day, the gland will harden and enlarge. Apparent cure can be obtained in 80% of these primary cases; but if after twelve to eighteen months' treatment a gland enlargement persists, it is wise for the patient to submit to operation to avoid possible relapse. Pathological examination of such glands generally shows that a good deal of hyperplasia still persists. At middle age one occasionally meets with a patient showing full clinical evidence of thyrotoxicosis, but without gland enlargement—the *forme frustre* of this complaint—and all such patients respond well to medical treatment.

Secondary thyrotoxicosis is a different problem, as the condition develops on an already abnormal gland—occasionally in the twenty to thirty years age period on an old colloid thyroid, or more commonly in the thirty-five to fifty years age group on multiple colloid adenomata, or occasionally on the single adenoma. In these patients toxicity comes under control rather rapidly; but all require operation to effect permanent cure. The best time to operate is some four to six months from the commencement of treatment, as by then the patients' general physical condition has picked up and they do not worry about the operation. If operation is carried out earlier there is usually considerable perithyroiditis, which makes the stripping of fascial planes difficult, and the vascularity of the gland has not subsided. Both these conditions increase operative difficulties very much, with resultant slowing up of the patient's convalescence.

Method of Treatment.

As a result of the continuous stimulation of function of all body tissues from the excess thyroxine production, the patient develops an asthenia of both nervous and muscular systems, so that their rehabilitation is just as essential as is control of the hyperfunctioning gland. Do not forget that pituitary activity is part of the process too, and that some reduction of stimuli from cerebral cortex to basal ganglia and thence to pituitary is necessary. The patient should be encouraged to make every effort to reduce worry and stress and to make an adjustment to any psychological factors. The barbiturates seem to give best results, with

[1] Read at the fourth annual post-graduate course of the New South Wales Federation of Country Local Associations, Wollongong, March 14 to 18, 1955.

[2] It was listed on June 1, 1955.

chloral hydrate at bed-time if sleeplessness is part of the picture. Adequate dosage must be given to secure control, and as the patient improves the doses can be reduced. The *B* group vitamins are best to restore tissue tone, especially if they are combined with glycerophosphates. If there has been much weight loss, "Milo", "Bournvita" or "AktaVite" may be used as a food supplement. These ancillary measures must be carried on practically through the whole period of treatment.

With regard to the antithyroid drugs, both methyl thiouracil and propyl thiouracil require comparable doses, although experimentally the propyl compound was thought to be more efficacious than the methyl compound; but clinical experience shows that this does not hold in human beings. It is no use giving small doses to start with, as control of toxicity is too slow and gland hyperplasia will be increased. For the average patient 0·1 gramme should be given three times a day for one week, then the dose should be doubled for the second and third weeks; by this time the break in thyroxine production has been achieved and the excess in circulation has been eliminated. The dose can then be reduced to 0·1 gramme three times a day, as this will maintain a satisfactory balance; usually at the end of six weeks the favourable clinical response is evidenced by a sense of well-being, increase in weight, loss of tremor and tachycardia *et cetera*. If this "adequate response" has been achieved, the dose can be reduced to 0·1 gramme twice a day, and if the gland is very large the administration of *Thyroideum Siccum* may be commenced, in a dosage of 0·5 gramme twice a day, as this helps to balance pituitary output of thyrotropic hormone and results in diminution of gland hyperplasia. If the thiouracil is not balanced in this way with *Thyroideum Siccum* it will increase the hyperplasia due to the uncontrolled thyrotropin, and also exophthalmos will increase. After another four weeks the dosage of thiouracil can be further reduced to 0·05 gramme twice a day with 0·5 gramme of dry thyroid as maintenance dosage for some three to six months; after this the dose of thiouracil can be further reduced to 0·025 gramme twice a day, still with dry thyroid for a further three or four months at least. No matter how well the patient seems, it is unwise to cease all treatment in less than twelve to fifteen months, otherwise relapse may occur. At the commencement of treatment the patient should be examined once a fortnight and then at the intervals when change of dosage is due; but in the "maintenance stages" once in two to three months should suffice. If this method of dosage is followed, it is seldom that any toxic reactions occur. If "Neo-Mercazole" is used, it should be remembered that this is put up in five-milligramme tablets. As it does not produce any toxic reaction or gland hyperplasia, the initial dosage of two tablets three times a day can be given, or in very severe cases three tablets three times a day for a week or two; then the dose may be gradually reduced as improvement occurs, but the maintenance dosage should be not less than one tablet twice a day. Supplementary dosage with *Thyroideum Siccum* is required if the gland is enlarged. As with most other drugs, the best and safest dose of the antithyroid substances to use is the smallest dose that will give the clinical result desired.

Preparation for Operation.

For those patients with primary thyrotoxicosis who have a persistently enlarged gland which will not recede after nine to twelve months' treatment operation is advisable. It is also advisable for the majority of patients who have thyrotoxicosis classed as secondary.

Operation should not be rushed in any case; it should be planned deliberately when the patient has regained a relatively normal level which has been stable for at least two months. The mortality now in experienced hands is about 1 in 1500, and morbidity can be greatly reduced when in the operative field the fascial planes strip easily and vascularity of the gland has abated. Absolute hæmostasis is an essential, as also is efficient anæsthesia.

When the thiouracils are being given, these should be discontinued fourteen days prior to operation, being replaced by Lugol's solution in a dosage of three minims twice a day. If larger doses are given or if the time is extended, iodine thyroiditis and perithyroiditis develop and make technique more difficult. "Neo-Mercazole" may be used right up to the day of operation and is best for patients susceptible to iodine in any form; but when it can be taken it should be used for ten to fourteen days prior to operation. If the patient has been adequately prepared, there should be no thyroid crisis or other post-operative reaction. Since the routine aspiration of the trachea, main bronchi and pharynx before the patient leaves the operating theatre has been adopted, post-operative lung complications seldom arise.

Toxic Reactions.

Experience has shown that it is unnecessary to have repeated leucocyte counts made. It is just as well to have an initial count made, as individuals vary greatly, and if the number is 4000 per cubic millimetre or below with a total granulocyte count of 40% or less, it is wise to reduce the commencing dosage by half and warn the patient to stop treatment at once if any fever or sore throat arises, and to report without delay. A leucocyte count should be made, and if leucopenia occurs pyridoxin should be given and thiouracil stopped for three or four days, when its administration may be resumed in half-doses. Agranulocytosis is a rarity if the dosages recommended are followed. If it does occur, penicillin should be given until daily counts show resumption of bone marrow activity. "Neo-Mercazole" should then be substituted for the thiouracil, and usually operation is advisable a little later on. Occasional joint pains may occur, which yield to aspirin; rarely toxic erythema or a mild febrile reaction may be met with, both of which clear when treatment is stopped for forty-eight hours and resumed with reduced dosage, or when a change is made to another preparation of antithyroid drug.

Thyrotoxicosis in Pregnancy.

Pregnancy may occur during treatment, or thyrotoxicosis may first develop during pregnancy. In neither case is it necessary to terminate this physiological state, as treatment may be carried out along the approved lines up to the end of the seventh month. At that time thiouracil should be suspended and replaced by two or three minims of Lugol's solution, given twice a day, until the child arrives and for three months afterwards. This will prevent any hyperplasia of the fœtal thyroid, and it keeps the mother's thyroid on a stable level. It is not generally known or appreciated that in the last six or eight weeks of pregnancy the mother's basal metabolic rate rises to between +15% and +25%, but drops immediately on the birth of the child. If operation is considered desirable it is best carried out at about the fourth month, but can be safely performed up to the sixth month; after that the risk to both mother and child begins to rise.

Conclusion.

In conclusion I would advise a common-sense approach to the problem of thyrotoxicosis. Diagnosis must be certain, and treatment, both ancillary and definitive, must be carried out regularly along approved lines for a long period. The patient should not be worried with repeated basal metabolic rate estimations or leucocyte counts. The dosage should not be altered every few weeks or hypothyroidism may easily be induced with too large doses over long periods.

Finally, I would advise you to study each patient as a separate problem, to rely on your own clinical acumen in estimating the progress, and, if there is any doubt, not to hesitate to ask advice from a more experienced colleague.

THE TREATMENT OF "DUSTED" MINERS AT THE WOLLONGONG PULMONARY CLINIC.[1]

By K. G. OUTHRED,
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A PULMONARY CLINIC has been established at the Wollongong District Hospital for the primary purpose of caring for members of the coal-mining industry who suffer from chronic non-tuberculous respiratory disease. It is our objective at this final session of the 1955 FOCLA post-graduate course to describe and demonstrate the work being done by the clinic and to give a brief indication of the results obtained to date.

In the main, the patients accepted for treatment are suffering from simple pneumonokoniosis, from chronic bronchitis, from emphysema, or from varying admixtures thereof. Our patients are referred by medical practitioners, our treatments are complementary to the ordinary therapeutic measures, most of which have already been tried by the patients' own doctors before they attend the clinic, and our objective is to assist as far as possible in the rehabilitation of lungs whose function is impaired, and in the prevention of further damage.

In the present initial stages of the clinic, patients in the above-mentioned classes are being treated largely on an experimental basis. Patients with bronchial asthma and bronchiectasis, whether mine workers or not, are accepted for treatment, mainly on routine lines.

Before describing the treatments used, I propose to make some brief remarks on the definitions of these diseases.

The term pneumonokoniosis, although redefined at the 1950 conference of the International Labour Organization, held at Sydney, as "a diagnosable disease of the lungs caused by the inhalation of solid particulate matter", is for the practical purposes of the clinic regarded as indicating fibrosis of the lungs of generalized nodular type, with or without massive lesions. Its presence in many cases in which there is no bronchitis or emphysema is demonstrable only by radiological methods, although the symptomatology may indicate some impairment of respiratory reserve, which is almost invariably confirmed by the pulmonary function tests.

For our purpose we define chronic bronchitis as the presence of chronic cough and wheeze, in sufficient degree to be troublesome to the patient—for example, by regularly disturbing his rest at night, or bad enough to have made him visit his doctor—and of at least six months' duration. There is usually present also an element of chronic bronchial obstruction ("miners' asthma") due to bronchospasm, to swelling of bronchial mucosa causing narrowing of the lumina, to plugging by inspissated secretion, to external pressure from enlarged glands, deformity or cicatricial stenosis of bronchi, or to a combination of two or more such causes.

Patients with recurrent acute or subacute bronchitis we accept for treatment as being probably in the preliminary stages of the more serious chronic condition.

Pulmonary emphysema was aptly and succinctly defined as "holes in the lung" by Dr. C. M. Fletcher, then Director of the Pneumonokoniosis Research Unit of the Medical Research Council of Great Britain, when he was in Sydney in 1950. The types of holes which develop in coal-miners are shown in the accompanying photographs (Figures I to IV) and whole lung sections which you see on display. These have been prepared from the lungs of New South Wales coal-miners obtained at post-mortem examinations, and are arranged in such order as to show clearly the progression of the disease.

The late Dr. C. H. Badham, then Director of Industrial Hygiene in New South Wales, in 1933 first drew attention to this truly occupational type of emphysema which develops in association with coal-dust pneumonokoniosis; and the technique for demonstrating its presence in post-mortem material by means of whole lung sections was evolved in the department of Professor Jethro Gough, of the University of Cardiff, South Wales, about 1948. However, not all emphysema is occupationally caused; but the clinical picture of emphysema appears to conform to certain patterns, whatever its cause.

FIGURE I.
Magnification 4.

FIGURE II.
Magnification 4.

Perhaps partly because the holes in emphysema throw no shadows in the X-ray film of the chest, there appears to be some reluctance to diagnose the presence of emphysema until it has developed to an advanced and hopelessly crippling stage. We have endeavoured to grade the disease into four stages of increasing severity and have set down

[1] Read at the fourth annual post-graduate course of the New South Wales Federation of Country Local Associations, Wollongong, March 14 to 18, 1955.

in some detail the criteria we use for diagnosis and grading.

A brief study of the prevalence of these chronic pulmonary diseases will convey to you the background of the establishment of the clinic. Although no reliable figures are available at present, it seems probable that there is a

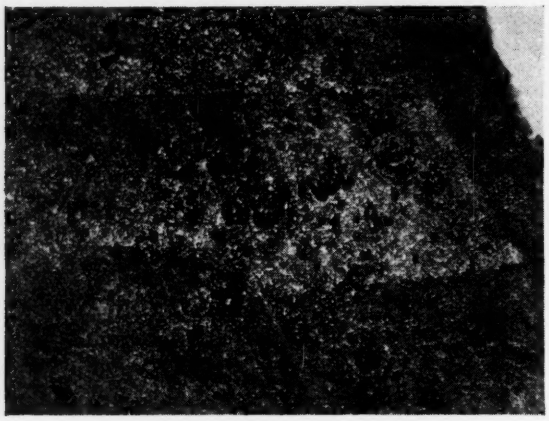

FIGURE III.
Magnification 4.

high incidence of such diseases in the general population of Greater Wollongong, and especially amongst workers in the heavy industries of the area.

In the coal-mining industry, George and Wallman, in 1948, having carried out medical examinations of a random

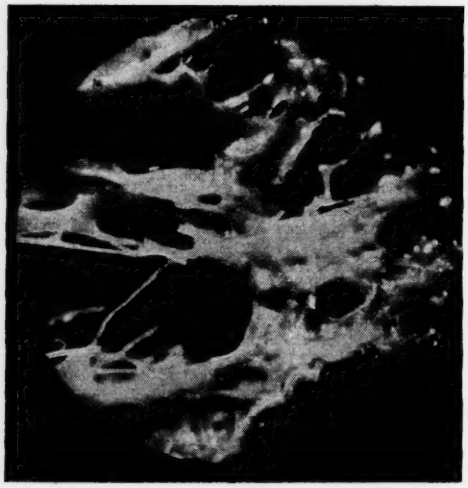

FIGURE IV.
Magnification 40.

10% sample of employees still at work, found the prevalence rates to be as follows: pneumonokoniosis (moderate to major degree), 3·4%; chronic bronchitis, 11%.

Routine medical examination, during the years from 1948 to 1952, of some 3125 men still employed in the mines, by the medical officers of the Joint Coal Board, has very closely confirmed the above figures. The results, expressed in overall prevalence of the commoner diseases only, are shown in Table I.

The incidence of pneumonokoniosis is largely confined to underground employees in the mines. Interestingly enough, analysis of the "chronic bronchitis and emphysema" cases reveals a fairly low incidence, not significantly different in the surface and underground workers, up to the age of forty years, and a steep rise after forty-five years, particularly amongst underground employees, the overall incidence being three times as high in the underground men as in those employed above ground (see Table II).

TABLE I.
Southern District Coal Field, New South Wales, 1948 to 1952: Prevalence of Respiratory Diseases.

Respiratory Disease Found.	Percentage of 3125 Miners Affected.
Pneumonokoniosis (moderate to major degree)	4·0
Chronic bronchitis, bronchial obstruction, emphysema	10·0
Pulmonary tuberculosis:	
Active	0·42
Arrested or healed	1·3

Such conditions as bronchial carcinoma, bronchiectasis and bronchial asthma are rarely for long compatible with continued employment in the mines, and no significant incidence of these diseases has been found amongst men still at work.

As a result of a widespread application of dust suppression measures in the collieries since 1945, a reduction in the incidence of pneumonokoniosis appears to be taking place, but the position in regard to bronchitis is not yet clear. Furthermore, although once again exact figures are not available, there is quite an appreciable legacy of these diseases amongst the older retired mine-workers in the district.

TABLE II.
Chronic Bronchitis, Bronchial Obstruction, Emphysema: Prevalence Amongst 1300 Surface and Underground Employees in Broad Age Groups.

Type of Employee.	Age Group (Years).		
	Under 45.	45 and Over.	All.
Surface	1·4%	4·2%	2·6%
Underground	2·8%	14·1%	8·2%
All	2·5%	12·6%	7·2%

With this background of disease prevalence in view, the Wollongong Pulmonary Clinic was established to provide organized facilities for investigation and treatment of chronic diseases other than tuberculosis. Whilst the clinic accepts for treatment patients suffering from bronchiectasis and bronchial asthma, the primary purpose of the clinic, in its present initial stage, is to evaluate certain methods of treatment of "dusted" and bronchitic miners in regard to which favourable reports have been appearing in the literature of the past ten years. A secondary aim is the study of whole lung function testing, with an ultimate objective of selection of one or more tests as suitable for routine use in the prevention programme.

In the industrial and mining centres of the United States claims have been made of the value in these diseases of positive-pressure breathing combined with aerosols. Motley in Pennsylvania and Segal in Boston recommended oxygen, physiotherapy, detergents and positive-pressure breathing, as well as various combinations of drugs. It seems that in London and Wales there is scepticism regarding the use of positive-pressure breathing machines, and the emphasis is on physiotherapy, with aerosols and other well-tried methods of treatment. Scadding holds that with physiotherapy the diaphragm can be made to move like a piston.

However, Donald has stated that physiotherapy has no real place in treatment.

The two main methods of treatment being investigated at this clinic, then, are (i) physiotherapy and (ii) intermittent positive-pressure breathing.

Of the patients referred to the clinic, those suffering from pneumonokoniosis, chronic or recurrent bronchitis, and/or emphysema, without significant other disease, have been selected for treatment by these methods. The presence of uncomplicated arteriosclerosis or mild grades of hypertension has not been held to exclude any individuals from selection. Up to the present time, the great majority of such patients who have presented for treatment have been retired mine-workers within the age group fifty to seventy-five years. A few are still employed in the mines or in other heavy industries in the Port Kembla area.

A small percentage of patients have associated chronic or subacute upper respiratory tract disease. When it is present, such disease is energetically treated in addition to the pulmonary condition.

In passing, it must be stated that, because of an extreme shortage of available hospital beds, only a few of the most urgently ill patients have been able to receive in-patient treatment. The majority of patients with disease of all grades of severity have been treated entirely as out-patients.

Clinical assessment of all patients selected has been used, together with radiological, fluoroscopic and electrocardiographic examination, both in diagnosis and in grading of the severity of disease. Respiratory function tests have also been used to classify the degree of pulmonary disability, and in our first group of patients there has been found to be a good correlation between the clinical assessment and the grading as determined by function tests.

Reassessment by clinical methods and by function testing is carried out in each case after a course of treatment.

The treatment given has been briefly as follows:

1. Physiotherapy. A course of training in breathing and relaxing exercises was given to each patient. Individual tuition was given for two periods of thirty minutes each, and then the patients were grouped into classes of six and were given daily exercises for two weeks. They were then given class training twice a week for four weeks, and thereafter weekly or monthly, or as often as appeared individually necessary.

2. Intermittent positive-pressure breathing. Each patient was given ten to fifteen minutes' breathing twice per day at a gradually increasing pressure, up to 20 cubic millimetres of water. Individual patients with bronchospasm were also given a bronchodilator with the oxygen during positive pressure breathing. In addition, each patient was given oral inhalation of detergent for ten minutes each hour. This resulted in each patient having daily five or six periods of oxygen breathing, two under pressure and three or four by simple inhalation. The course of treatment with oxygen usually extended over a two-week period, but in some cases was carried out for up to six weeks. The detergents used have been one in 1000 aqueous "Zephiran" solution and "Alevaire".

3. Medical treatment. Most of these patients were under treatment by their own doctors with various prescriptions. However, a few were not receiving any medicaments, and in some cases their own doctors were asked to give certain prescriptions such as "Benadryl", iodides or antispasmodics, or in an intensive course of antimicrobial therapy. However, the use of such measures was kept to a minimum, so that if possible the effect of other treatment could be determined.

Conclusion.

The results of the work of the clinic to date tend to confirm reports from elsewhere—that much can be done to improve these chronically ill patients and alleviate their symptoms, even in the more advanced stages of disease. It is believed that intensive treatment, in the earlier stages and of acute infective episodes, may do much to prevent the development of the more severe irreversible, obstructive lesions and of emphysema of the lungs, or at least help to retard their further progress. Efforts are now being made to encourage patients with early recurrences to report for treatment during the acute phases of their illness.

Acknowledgement.

The Joint Coal Board has given permission for the publication of this paper.

ASPECTS OF DIAGNOSIS IN CHRONIC BRONCHITIS AND EMPHYSEMA.[1]

By R. G. EPPS, M.R.C.P.,

Honorary Research Officer (Medical), The Royal North Shore Hospital of Sydney; Consultant Physician, Wollongong District Hospital Pulmonary Clinic.

MY purpose is to discuss the diagnosis and clinical assessment of severity of chronic bronchitis and emphysema and to refer to the experience gained at the Wollongong District Hospital's clinic in the study of these diseases.

Hypertrophic vesicular emphysema or "large-lunged" emphysema has been frequently found amongst the patients attending the clinic and the clinical diagnosis confirmed by pulmonary function studies. Chronic bronchitis and pneumonokoniosis have been found to be the responsible ætiological agents in most cases, but in a few instances bronchial asthma was responsible.

The diagnosis of chronic bronchitis must depend largely upon the history of recurring cough, wheezing, and frothy, sometimes mucopurulent sputum, becoming worse in the winter and following regularly after bad colds. The finding of inspiratory and expiratory rhonchi and basal râles in the chest is helpful confirmatory evidence, but is not always possible during remissions and is not essential for diagnosis. The same remarks apply to the assessment of severity of chronic bronchitis, and this must be based mainly on the patient's history.

The diagnosis and assessment of severity of hypertrophic emphysema rest mainly upon the physical and fluoroscopic examination of the chest; but several points in relation to the history are worth mentioning.

Symptoms and Signs.

Dyspnœa on effort is always present and may vary in degree from mild to severe, in the worst cases being present at rest; when this is so, caution is necessary in submitting the patient to tests of pulmonary function, for a succession of quick deep breaths will aggravate the dyspnœa and may produce faintness or even a syncopal attack. Dyspnœa is aggravated by heart disease, an anxiety state or other factors. Cough, wheezing, expectoration and tightness in the chest are common symptoms, and sharp pains in the chest and shoulders, presumably of muscular origin, are often complained of.

Orthopnœa, dizziness and fatigue are not uncommon. A striking loss of weight and a dulling of the mental faculties are common features in the severe cases.

The time is too short for me to discuss all the physical signs of emphysema that are described, and many are unimportant; but I should like to refer to a few which have been found the most reliable, and to several which are widely accepted but may be misleading.

The basic abnormalities which produce the physical signs are an increase in volume and loss of elasticity of the affected lungs.

[1] Read at the fourth annual post-graduate course of the New South Wales Federation of Country Local Associations, Wollongong, March 14 to 18, 1955.

The over-expanded, almost immobile barrel chest is seen in some instances of gross emphysema, but more often the chest movement is only moderately restricted (to an expansion of about two inches) and yet considerable emphysema is present.

The movement and position of the chest wall and the diaphragm must both be considered, for either or both may be affected, and in some instances the thoracic cage appears normal and moves normally, but the diaphragm is depressed and immobile owing to considerable emphysema. Fluoroscopic examination is necessary for accurate assessment of movement and position of the diaphragm.

Increase of resonance on percussion over the chest is a helpful sign, when allowance is made for the thickness of the patient's chest wall, but is sometimes not appreciable in milder degrees of emphysema.

The most valuable physical signs are the decrease in or absence of cardiac and liver dullness and the decrease in intensity of the heart sounds, or their absence at the base, and often also the apex of the heart.

Dullness on percussion over the precordium, with the patient reclining and breathing quietly, is a constant finding in health, and its impairment is reliable evidence of emphysema, provided that the mediastinum is not grossly displaced. The area of liver dullness is less constant in health, and consequently its loss in emphysema is rather less reliable.

The heart sounds are heard best at the lower end of the sternum in emphysema, and the fact that they are poorly heard or absent at the base of the heart, and often also at the apex, is good evidence of its presence. This sign is due to the intervention of emphysematous lung tissue between the chest wall and heart, and not to myocardial damage, as is often suggested.

Central cyanosis is a much over-emphasized sign in emphysema, and was present in only a few of the many patients examined. Clubbing of the fingers was only occasionally seen.

Chronic *cor pulmonale* is a common late complication of emphysema, but was only occasionally encountered in the group of out-patients studied.

Radiological Signs.

In cases of gross emphysema the postero-anterior X-ray film of the chest may show a low, flat diaphragm, increased translucency of the lung fields with diminished lung markings, a small central vertical heart shadow, and horizontally placed ribs with wide intercostal spaces. When lesser degrees of emphysema are present, the plain chest X ray is likely to appear normal or very nearly so, and to be of no value in diagnosis.

The lateral X-ray film of the chest may add further information by showing an increased antero-posterior diameter and increased areas of the retrosternal and retrocardiac spaces.

Fluoroscopy.

Fluoroscopy is the most essential examination, for with it diminished movement of the diaphragm can be appreciated. In health, both leaves of the diaphragm move equally from two to four inches between full inspiration and full expiration, and in emphysema the range of movement is greatly restricted or even partly paradoxical. The appreciation of this abnormality is important not only in diagnosis and assessment, but also in treatment, for it can usually be corrected with very beneficial results.

Electrocardiogram.

The electrocardiogram may be normal in emphysema, but the electrical axis of the heart tends to become more vertical, and clockwise rotation of the heart about its longitudinal axis is common. These changes appear to result from the descent of the diaphragm, and produce right axis deviation in standard leads, a vertical heart position in unipolar limb leads, and a right ventricular *RS* pattern in the chest leads to V_5 or V_6. These features are not usually found in a heavily built, normal subject, whose chest is broad, and when they are found in this type of patient strong evidence in favour of emphysema is available.

It so happens that many patients attending the clinic with mild or moderate degrees of emphysema have had this sthenic type of body build, and the electrocardiogram has given support to the diagnosis by showing these changes. In a thin, tall person such changes are of little significance and do not help.

When more severe emphysema is present a low voltage *QRS* complex is likely to be found in the standard leads and also in the left ventricular surface leads.

Evidence of associated chronic pulmonary heart disease is found in the presence of tall *P* waves in lead II, of amplitude greater than 1·5 millimetres, with or without evidence of right ventricular hypertrophy or strain in the chest leads.

Conclusion.

In conclusion, it has been possible to grade the severity of emphysema, on the results of physical, fluoroscopic and electrocardiographic examination, into four categories, the first being the mildest and the fourth the most severe, and we have been impressed with the close correlation found between the assessment arrived at on clinical grounds and that found by respiratory function tests.

WOLLONGONG PULMONARY CLINIC: PULMONARY FUNCTION TESTING.[1]

By M. J. FLYNN,
Medical Officer, Joint Coal Board, Sydney.

PULMONARY function tests will not disclose the site, the size, the nature or even the existence of a lesion if that lesion is not sufficiently extensive to interfere with lung function. Miners with extensive radiological evidence of pneumonokoniosis may have little disturbance in function, while others with minimal radiological evidence of disease may have true pulmonary disability.

In this clinic respiratory tests are used to determine the extent of disability, (i) as a diagnostic aid, (ii) as a measure of the effectiveness of therapy, (iii) as a study of the progress of the disease.

Methods.

Respirometer Studies.

The patient breathes into a respirometer, from which a tracing is obtained of his respiration at rest and during maximal effort. These tracings form a permanent record from which may be obtained an analysis of the breathing pattern and various measurements of lung ventilation. Of particular value are the maximum breathing capacity, the vital capacity, the combined vital capacity and the timed vital capacity.

The best test at present is the estimation of the maximum breathing capacity, which is the maximal volume of air breathed in unit time expressed as litres per minute. The patient's ventilation is measured over a period of twelve to fifteen seconds, during which the patient is continually urged to make his maximum respiratory effort. Dyspnœa is more closely related to reduction in maximum breathing capacity than in the vital capacity.

The vital capacity is the volume of gas expelled by maximal expiration following a maximal inspiration or vice versa. The vital capacity on its own has a very limited value, but variations exceeding 250 cubic centimetres have some significance as a measure of progress.

[1] Read at the fourth annual post-graduate course of the New South Wales Federation of Country Local Associations, Wollongong, March 14 to 18, 1955.

Combined vital capacity is the sum of the inspiratory capacity and the expiratory reserve volume measured separately. If the combined vital capacity is smaller than the single measurement of vital capacity, it is evident that the subject is not fully cooperating. In asthmatic and emphysematous patients the combined vital capacity may exceed the single vital capacity by as much as one litre.

The timed vital capacity is the maximal expiratory volume recorded in seconds as fractions of the total vital capacity. This test quickly detects any obstruction to breathing. Asthmatic and emphysematous patients can record high vital capacity if given sufficient time; but in one or two seconds they may be able to expel less than half their total capacity.

TABLE I.
Evaluation Standards.

Lung Function Test.	Degree of Lung Function Impairment: Grade				
	0 None.	1 Slight.	2 Moderate.	3 Severe.	4 Advanced.
Maximum breathing capacity — percentage of that predicted	90+	80–90	55–80	35–55	35–
Timed vital capacity:					
One second (percentage of timed vital capacity)	80+	60–80	50–60	30–50	30–
Three seconds (percentage of timed vital capacity)	95+	80–95	60–80	40–60	40–
Vital capacity: percentage of that predicted ..	90%	70–80	60–70	40–60	40–
Combined vital capacity ..	±	±	+	+	+ + (1 litre) +
Emphysema: pattern of maximum breathing capacity:					
(i) Staircase phenomenon				++	++
(ii) High mid-capacity ..			+	++	++
Dyspnœa time with step-up exercise (seconds) ..	90–	90–120	120–180	180–300	300+

In bronchitis and emphysema these tests show a pronounced reduction from normal figures, which can be predicted from formulæ which take into consideration the patient's weight, height and age.

The instruments used in these tests are the Collins 13·5 litre respirometer and the Gaensler timed vitalometer. Studies of the tracings may also reveal respiratory incoordination, bronchospasm and air trapping.

Exercise Tests.

The standard American exercise test is that of Cournand and Richards, in which the patient ascends 30 eight-inch steps in one minute. Measurements are made of the patient's pulse and respiration and the time of his dyspnœa.

The standardized exercise test devised by the Pneumonokoniosis Research Unit in England takes consideration of the patient's build, and makes the rate of stepping and the height of the step inversely proportional to his weight.

Types of Patients Tested.

Tests have been conducted on both healthy and sick miners, on hospital staff and on patients with asthma, bronchiectasis or other chest disorders. The patients have tests before, during and at the completion of therapy, and at subsequent periods thereafter.

Results of Tests.

Analysis of the test results shows an impairment of lung function which agrees very closely with the clinical assessment. The disability is divided into four grades, based on evaluation standards prepared by Motley and his co-workers in Pennsylvania, by Cournand and Richards in New York, and by the Pneumonokoniosis Research Unit in England.

Of the first 100 patients tested, the respiratory function grading was identical with clinical assessment in 70, and within 25% correlation in the remainder.

Results of Treatment.

Subjective Findings.

Each patient was asked whether he felt any change in his general condition and in what way he had noticed it. Improvement noted was generally in the reduction of cough, especially during the day and at night, in ease of expectoration and in improvement in exercise tolerance.

Objective Findings.

Patients were examined clinically with special regard to change in chest expansion, in respiratory movement and in the usual findings on auscultation.

Fluoroscopic examination was carried out with special regard to diaphragmatic movement.

In respiratory function tests the measure of improvement was an improvement of at least 10% in function testing.

Interim Results.

In the first six-month period of trial of combined physiotherapy and oxygen treatment 103 patients were selected for treatment. Of these, 45 showed subjective improvement without any measurable objective change; 35 made a subjective and objective improvement; one made an objective improvement without any subjective change; and four made a subjective improvement with deterioration in objective measurements. Seven showed no change in either subjective or objective measurements, and eleven were still under treatment and awaiting assessment.

Acknowledgement.

The Joint Coal Board has given permission for the publication of this paper.

A THYROID BLOCKING AGENT AS A CAUSE OF ENDEMIC GOITRE IN TASMANIA: PRELIMINARY COMMUNICATION.

By F. W. CLEMENTS,
Institute of Child Health,[1] University of Sydney, Sydney.

ALTHOUGH most authorities now agree that a deficiency of iodine in the food is a cause of endemic goitre, some of these authorities also join the minority in supporting the suggestion that there may be other ætiological factors in endemic goitre. Greenwald (1950), without much positive evidence, steadfastly challenged the iodine-deficiency theory. McCarrison (1928), while conceding that iodine might play some part in goitre prevention, maintained that the ultimate cause was a positive toxic agent derived from the gastro-intestinal tract. Astwood (1949) has suggested that the consumption of foods containing substances which could be converted in the body into thiocyanates would render iodine-deficient a diet which otherwise would be adequate in iodine, and that this might explain the fact that endemic goitre exists in regions of the world where the iodine of the soil and water is abundant. More recently De Smet is quoted ("Goiter in the Congo", 1954) as believing that in addition to eating a diet deficient in iodine, many victims of endemic goitre in the Belgian Congo ingest a "certain quantity of 'blocking bodies' which are quite plentiful in cassava".

The results of two goitre surveys in Tasmania at an interval of five years, and subsequent investigations, strongly support the hypothesis that a significant amount of the endemic goitre in that State is due to the "blocking" effect of a substance or substances present in the diet of

[1] Endowed by the Commonwealth Department of Health.

children. This is a preliminary report of some of the findings; more detailed information will be given in later publications.

After a goitre survey in 1949 of some 8000 school-children, which confirmed that most of the island was goitrous, tablets containing 10 milligrammes of potassium iodide were distributed weekly to school-children in Tasmania for the next five years. This method of goitre prophylaxis had been found to be effective in another goitrous area in Australia—namely, the city of Canberra (Clements, 1953). It is conceded that the distribution of the tablets at a number of schools in Tasmania was irregular and unsatisfactory, but at most schools it was satisfactory. The second survey in 1954 showed a fall in the incidence of goitre in children of all ages in two districts, and in older girls in most districts; but there was a considerable rise in incidence in the younger children in most of the island, and the rate for boys equalled that for girls. This increase had occurred even in those schools where the distribution of iodine tablets was regular and complete. The results of the second survey confirmed that a significant part of the goitre in Tasmania was due to a deficiency of iodine in the diet; but they also suggested that a considerable amount might be due to another factor.

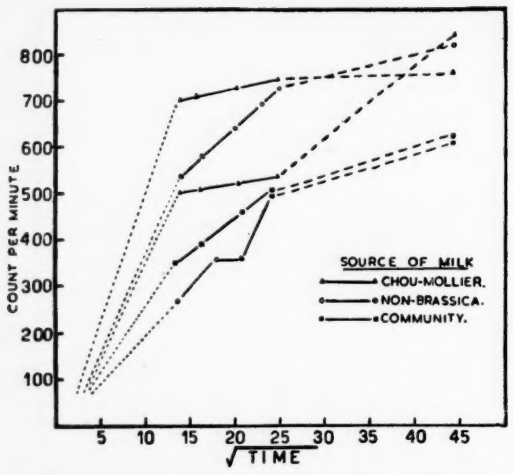

FIGURE I.

Uptake graphs of radioactive iodine (10 microcuries) administered at zero time to five girls, aged ten years. Milk, as indicated, substituted for normal milk in the diet. Approximately two and a half pints of milk consumed between 8 a.m. on the day of the test and 9 a.m. the following day. Last reading obtained at 11 a.m. on the second day (45 on the chart). The curves of uptake of I^{131} of the two girls who consumed milk from cows fed chou-mollier showed considerable interference with iodine uptake compared with the controls.

In the five years between the surveys there was a considerable increase in milk consumption by children in Tasmania, owing to the introduction of the Commonwealth Government's free milk scheme. To meet the demand for extra milk a number of changes were made in dairy practice, including a great increase in the production of a forage crop, chou-mollier (thousand-headed kale), which is one of the Brassicæ. In view of the discovery by Astwood and his co-workers (Greer and Astwood, 1948) that some Brassicæ contain an antithyroid substance—namely, *l*-5-vinyl 2-thiooazolidone—it was decided to test for antithyroid activity milk, and extracts from milk, from cows fed chou-mollier and other Brassicæ. Through the cooperation of Dr. H. M. L. Murray, of the Department of Public Health, Tasmania, of Mr. H. E. Hall, Government Analyst, Tasmania, and of officers of the dairy section of the Department of Agriculture, samples of milk and various extraction fractions of milk have been obtained. Samples of milk from herds at the Hawkesbury Agricultural College, New South Wales, have been used for control tests.

The tests have been made initially in human volunteers by the use of the technique described by Greer and Astwood. The subjects were given a small dose (20 microcuries for adults and 10 microcuries for children) of I^{131}, and the rate of its accumulation by the thyroid gland was recorded with the scintillometer at the Unit of Clinical Investigation, Royal North Shore Hospital, which was placed at our disposal by the Director, Dr. F. F. Rundle.

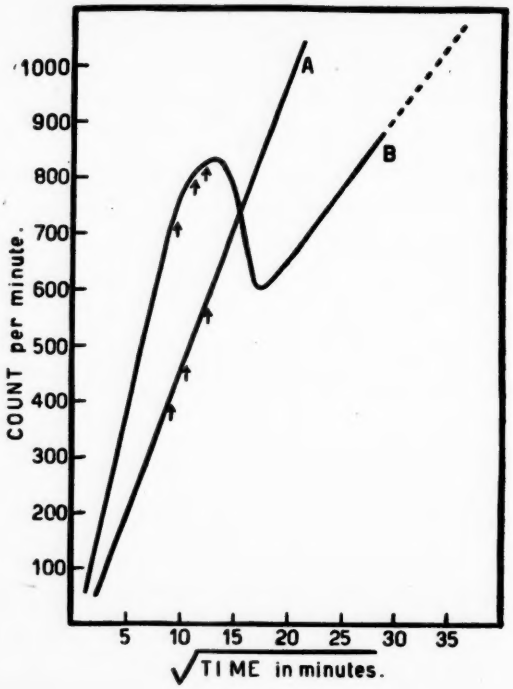

FIGURE II.

Uptake curves of radioactive iodine, showing effects of different samples of milk. A = control milk. B = chou-mollier-fed milk.

The results of two experiments are given in the accompanying graphs. Figure I shows the accumulation of I^{131} in five girls, aged ten years, who were given the I^{131} at zero time (7 a.m.), and who consumed approximately one and a half pints of milk. This was substituted for the ordinary milk in their diet at meal times, and at mid-morning and mid-afternoon. A further half-pint was consumed on the morning of the second day about two hours before the last reading at $\sqrt{T} = 39$. The two girls who drank the milk from cows fed chou-mollier had definite interference with the uptake of the I^{131} compared with the uptake of the other three girls. A pair of identical twins was used, one in each group.

Figure II shows the accumulation of I^{131} in the same subject in two tests at an interval of about one month. On both occasions a pint of milk was drunk at the times indicated by the arrows. One curve shows the steady accumulation of I^{131}, the other shows considerable interference, with some discharge of the I^{131}. This curve is similar to those regularly obtained in diagnostic tests with either propyl-thiouracil (Astwood, 1949) or "Neo-mercazole" (Macgregor and Miller, 1953). Similar results have been obtained in tests on other subjects with whole and skimmed milk, and to a lesser extent with various extracts.

While chou-mollier is a comparatively recent introduction into Tasmania, other Brassicæ have been used for winter feeding of dairy cows for many years. The investigation of milk from cows fed various Brassicæ is proceeding, and attempts are being made to obtain the antithyroid substance in a highly concentrated or pure form.

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Reports of Cases.

PULMONARY INFECTION IN SJÖGREN'S DISEASE (KERATOCONJUNCTIVITIS SICCA): A REPORT OF A FATAL CASE.

By S. C. WIGLEY AND J. B. EGAN.

From the Greenvale Sanatorium, Melbourne.

SINCE 1933, when Henrik Sjögren described the syndrome which bears his name, many important papers have discussed the minutiæ of the ocular and other manifestations of the condition. In consequence, an elaborate picture has been painted of the syndrome, which in its fully developed form is seen most frequently in women at, or about, the time of the menopause. It includes as major features lachrymal gland involvement, with *keratoconjunctivitis sicca*, *rhinitis sicca*, *pharyngitis sicca*, xerostomia, *laryngitis sicca* and parotid gland involvement. Irregularly occurring accompaniments include polyarthritis, skin changes such as scleroderma, Raynaud's phenomenon, hypochlorhydria and achlorhydria, changes in the differential white cell count, and alterations in the erythrocyte sedimentation rate. The florid form of the syndrome occurs less frequently than incomplete forms, and the condition is characterized in its clinical development by exacerbations and remissions. Ellman, Parkes Weber and Goodier have published a valuable account of the pathology of the condition.

The fatal issue to an infection of the lungs sustained by a patient suffering from Sjögren's disease, and some features of the course of the illness, are the subjects of this report.

Clinical Record.

The patient was a housewife, aged fifty-three years, who had suffered from Sjögren's disease for some ten years. Her father had suffered from tuberculosis, and one sister had died of septicæmia. Her five children were healthy, and there was no family history suggesting Sjögren's disease. Her medical history aside from that of the Sjögren's disease was unimportant.

In 1943, after an operation on the nasal sinuses, she noticed increasing deafness, and later dryness of the eyes and mouth. For eight years she had been using artificial tears to relieve the dryness of the eyes, and chewing and swallowing had become increasingly trying functions. Four years after the onset of the mouth dryness, she became affected by bilateral parotitis, which was treated variously by duct cannulation, penicillin administration, and finally by deep X-ray therapy. The parotid swellings subsided after this latter treatment, and did not recur. She complained also of Raynaud's phenomenon.

Despite the burdens of her affliction she had managed her home until October, 1952, when she was admitted to a general hospital in Melbourne. Here, after the usual investigations, she was given a course of treatment with adrenocorticotrophic hormone (ACTH) in an attempt to ameliorate the symptoms of the condition. A radiograph taken prior to this therapy showed clear lung fields. During the course of the treatment, incomplete symptomatic relief was obtained in that she had less frequent need of artificial tears, and she found that swallowing food was easier. However, she developed symptoms relevant to the lungs, and a radiograph showed evidence of right upper lobe lobar pneumonia, the presence of which was suspected on clinical grounds. The pneumonia developed in the face of a penicillin "cover" for the ACTH. This therapy was discontinued, and treatment was begun with chloramphenicol, and later with penicillin and chlortetracycline. She was discharged from the hospital in February, 1953. Subsequently she had a cough, but was unable to produce any sputum. In November, 1952, a test meal examination had shown a histamine-fast achlorhydria. The Mantoux skin test produced negative results with 1 in 1000 and in 1 in 100 old tuberculin.

She was admitted to the Greenvale Sanatorium in September, 1953, with a diagnosis of pulmonary tuberculosis. Two months prior to this she had suffered a bout of pneumonia, which appeared at first to respond to penicillin; but a relapse of the condition occurred. A radiograph was taken, and it was on the evidence of this film that admission to the sanatorium was sought.

There can be few more distressing sights for the physician than that of an ill patient with established Sjögren's disease. Our patient appeared prematurely aged, and had obviously lost weight. She had a constant dry cough, had frequent recourse to artificial tears, and moistened her mouth and throat repeatedly with water or other liquids. The conjunctiva was congested. She was edentulous, and the angles of the mouth were excoriated. The tongue was small, red, smooth, and dry, and the buccal mucosa was dry and atrophic. Other relevant signs were confined to the lungs. Chest movement was poor. The breath sounds were diminished at the base of the right lung posteriorly, and expiration was prolonged in the axillæ. Coarse and fine râles were heard throughout both lung fields, being most obvious over the lower lobe on the right side, and in the left axilla. The temperature was 100° F., the respirations numbered 24 per minute, and the pulse rate was 94 per minute. The erythrocyte sedimentation rate was seven millimetres in the first hour, and 16 millimetres in the second (Westergren, 200 millimetres). Radiographs of the chest, taken three weeks previously, showed elevation of the horizontal septum on the right side with shadows of varying size and density scattered through the upper zone of the lung field, and to a lesser degree in the lower part of the lung. The shadows were most pronounced in the axillary subsegments of the upper lobe. On the left side flocculent shadows lay in the lower half of the lung field, and excavation was apparent in the apex of the lower lobe. There was a minor shift of the mediastinum to the left.

On the evidence available to us at that time, it was thought that a presumptive diagnosis of pulmonary tuberculosis was justified, and treatment with streptomycin and paraaminosalicylic acid (PAS) was begun. This was done with some reservation, because the radiograph did not support this diagnosis unequivocally, and in fact it could as readily have suggested non-tuberculous suppuration with abscess formation. In addition there was no bacteriological confirmation of the diagnosis. Efforts were made to obtain some sputum, and eventually examination of a small amount of pus failed to disclose the presence of acid-fast bacilli. A skin test with 1 in 1000 old tuberculin gave a negative result. In view of these results, streptomycin and PAS therapy was stopped. Culture of the sputum provided the important clue to the ætiology; *Staphylococcus aureus* was grown, and drug sensitivity tests showed that the organism was sensitive to oxytetracycline, but insensitive to penicillin, chloramphenicol and streptomycin. Oxytetracycline was given for fifteen days.

During this period the general condition of the patient improved and some weight was gained, but in a short time she complained of frequency of micturition with dysuria, and examination of the urine disclosed many pus cells. *Proteus vulgaris* was grown, sensitive to chloramphenicol, and this drug was given for eight days, at the end of which time all evidence of the urinary tract infection had disappeared, and the patient felt well. A few crepitations could be heard at the lung bases, and a radiograph showed substantial clearing of the lung fields.

This respite was short-lived. The patient again became feverish (temperature 100° to 101° F.), and the cough became troublesome. In addition the basic symptoms of the condition were intensified, and peripheral joint pain became a feature of the illness. The fingers were not clubbed. Râles could be heard more extensively over the lung fields, and a radiograph showed extensive, rather ill-defined shadows throughout both lung fields, most pronounced at the bases of the lungs. Sputum culture yielded a strain of *Staph. aureus* sensitive to streptomycin, but insensitive to penicillin, chlortetracycline, oxytetracycline and chloramphenicol. In view of this development, and of the fact that some relief of the basic condition had been obtained twelve months previously with ACTH, it was thought that treatment with streptomycin might be potentiated by the concomitant use of cortisone. Accordingly, streptomycin was given with cortisone, and there was a substantial improvement in the patient's general state. The temperature fell, the cough became less troublesome, and the lung adventitiæ were reduced. The abnormal shadows in the radiograph cleared rapidly. After fourteen days another sputum specimen was obtained. In addition to a *Staph. aureus* sensitive to streptomycin, a hæmolytic streptococcus (Lancefield group A) was grown. This organism was sensitive to chlortetracycline, and this drug was given with the streptomycin and cortisone. Eleven days later the streptococcus had disappeared from the sputum, but a *Staph. aureus* was still present. This organism was sensitive to chloramphenicol, but resistant to penicillin, chlortetracycline, oxytetracycline and streptomycin. Chloramphenicol and cortisone were given for a further three weeks.

The patient was now in good health. She had gained weight, and had no cough, and the only abnormal sign in the lungs was an occasional râle at the base of the right lung posteriorly. A radiograph showed virtually clear lung fields with perhaps some prominence of the vascular markings at the base of the right lung. Sputum culture yielded a growth of normal flora including a few colonies of Monilia (not *Candida albicans*). The erythrocyte sedimentation rate was four millimetres in the first hour and eight millimetres in the second. The hæmoglobin value was 106%, and the total and differential white cell counts were normal. The serum electrolyte pattern was within normal limits. The patient was discharged from the sanatorium on December 24, 1953, fifteen weeks after her admission.

She was readmitted to the sanatorium in mid-February, 1954. During her stay at home she had suffered a series of short-lived febrile episodes associated with respiratory symptoms, and a prolonged bout of diarrhœa which was controlled eventually with phthalyl sulphathiazole. On her admission to the sanatorium she was in good spirits, but the physical signs in the lungs were similar to those on her first admission. No sputum could be obtained for examination. A radiograph showed coarsely reticulated shadows at the lung bases, the hilar shadows were prominent, and throughout the rest of the lung fields the vascular markings were increased.

In the absence of a bacteriological lead penicillin was given. Little clinical change was evident, and the physical signs were unchanged. For three weeks no drug therapy was given. Eventually sputum was obtained, and a *Staph. aureus* was grown. This organism was only slightly sensitive to streptomycin, and it was insensitive to penicillin, oxytetracycline and chloramphenicol. Erythromycin was then given to the patient. The course of treatment lasted for ten days, during which time little clinical change was evident, nor did any radiological change occur. A further specimen of sputum grew a *Staph. aureus* virtually insensitive to all antibiotics; but in addition a formidable growth of *C. albicans* was obtained. Antibiotic therapy was stopped, and potassium iodide was administered. For the next four weeks there was little change in the patient's state. She was feverish, and had an unproductive cough which did not distress her. However, as the malady progressed, the effect of the long illness became reflected in the patient's attitude. Whereas formerly she had on occasions been jaunty and unconcerned, she now appeared melancholy, querulous and indifferent, presenting a trial and a challenge to all concerned with her well-being.

This hapless woman's terminal illness began over a short period with explosive violence. On May 16 she complained of pain in the peripheral joints, and shortly after became desperately ill with high fever, central cyanosis and the peripheral signs of a hyperkinetic circulation. Expiration was prolonged over the lung bases, and râles could be heard over a wide extent in the lung fields. It was evident that the pulmonary condition was wildly out of hand. A small amount of blood-stained sputum was produced. Penicillin in high dosage was given, and oxygen; but it rapidly became apparent that the clinical state was deteriorating. Purpuric hæmorrhages appeared over the malar regions, and five days after the onset of the illness a basal pericardial friction rub was heard, and this later became audible over the whole of the precordium. Death occurred on May 22. The specimen of sputum obtained before death yielded a growth of *Staph. aureus* only slightly sensitive to erythromycin, and insensitive to all other antibiotics. A few colonies of *C. albicans* were grown.

Necropsy Findings.

At necropsy the body of this middle-aged woman showed some peripheral œdema, malar purpura and bruising at the sites of injections. The liver and spleen were congested, ascites was moderately severe, and a small amount of fluid was present in the pericardial sac. Fibrinous pericarditis was apparent. On macroscopic examination no lachrymal or parotid tissue could be identified positively, and indeed histologically no lachrymal tissue was found, remnants of parotid tissue only being identified. Other organs with the exception of the heart, lungs and some mediastinal glands appeared normal to the naked eye. The lower lobes of the lungs were reddish, and quite firm. Pus could be freely expressed from the bronchi, and from other points on the cut surface. The middle lobe and the upper lobes were waterlogged. The mediastinal glands were swollen and purple in colour. Examination of sections of the "carneous" lower lobes revealed an organizing pneumonia of severe grade. Congestion was severe, and cell accumulations in many areas achieved the proportions of abscess formation. Many of the bronchi showed epithelial denudation, and in other instances stratification of the epithelium was apparent. Sections from other parts of the lungs showed the familiar pattern of pulmonary œdema. Sections from the larger bronchi and trachea showed a reduction in the number of submucous glands, and the survivors, effete in appearance, were the focal points of round-cell infiltration which was less obvious elsewhere in the submucosal tissues. Our histological survey was less complete than the exhaustive study, already referred to, of Ellman, Parkes Weber and Goodier; but the findings in sections from the conjunctiva, parotid gland, skin, buccal mucosa and other parts of the gastro-intestinal tract differed in no essential way from the full descriptions of similar tissues reported in their case. Other visceral sections were normal histologically.

Comment.

In 1951, Ellman, Parkes Weber and Goodier published a valuable account of an extensive pathological investigation of a patient who had succumbed to bronchopneumonia. This patient had also suffered from Sjögren's disease. They concluded, as a result of their study, that the basic pathological process in this disease was a chronic inflammatory atrophy of unknown ætiology, involving the lachrymal,

salivary, mucus-secreting and sweat glands, and leading to a pathological dryness of mucous surfaces.

Pathological dryness is an outstanding feature of the examination of the eyes, nose, mouth, pharynx and bronchial tree. The dryness of the mouth predisposes to infection of the parotid and other salivary glands, with the sequelæ commonly described as occurring in the condition. It seems clear that the unusual dryness of the respiratory tract will result in a grave depression of the natural clearing mechanisms of the lungs—namely, the secretion of mucus and the ciliary action of the respiratory mucous membrane—and a considerable strain must be placed on the local defences against bacterial aggression at the periphery of the bronchial tree. In view of this apparent susceptibility to infection of the lungs, it is of interest that there are so few reports of pulmonary complications in this condition. If the clearing mechanisms are nullified in this way, it might be expected that infection would readily become established in the lungs, and, once established, the nature of the pathological process would render eradication of the infection a difficult if not impossible task. It is possible that in the commoner incomplete forms of the condition these clearing mechanisms, even if depressed to some extent, are sufficiently effective to maintain the cleanliness of the bronchial tree in its lower reaches. Alternatively, in all forms of the disease, the local humoral and cellular defences can deal with infecting agents presented to them in ordinary circumstances. However, these defences may be breached if the development of infection is facilitated in any way. In our case the initial infection occurred during the course of treatment of the Sjögren's disease with ACTH.

There is ample evidence to indicate that the evolution of bacterial infection can be enhanced by ACTH and cortisone therapy. Such therapy appears to deprive the host of protection against unrestricted bacterial multiplication and invasion. The way in which it does so is not clear. That the drugs may act by depressing local cellular and humoral defences, or by interfering with tissue permeability, are possibilities for which there is some evidence. In a discussion of the relationship of cortisone and infection, Thomas has suggested that this drug may have a direct effect on the cells of the reticulo-endothelial system in the direction of the host's disadvantage. He has offered the theory that "the effect of cortisone is to interfere with the function of the R.E. system impairing its normal capacity to remove, fix, or detoxify bacteria and certain bacterial products. The result may be a shifting of these functions to tissues not normally concerned with them, with uncontrolled infection or tissue damage as the outcome". The organisms responsible for disease induced in this way may be the usual pathogens, or organisms normally harmless and occurring naturally on body surfaces, but made noxious to the host because of some disturbance in bacterial ecology.

The development of infection under cover of penicillin is notable. In ordinary circumstances the efficiency of an antibiotic drug is not interfered with by ACTH or cortisone, the reason probably being that doses of the drugs are commonly far in excess of the minimal requirements for effective control of infection. However, in the case of organisms resistant to a drug, the host is entirely dependent on natural defences, and a suppression of these by ACTH or cortisone (occurring not necessarily with excessive doses of these drugs, but in the range of normal therapeutic doses), may have a disastrous influence on the outcome of the infection. Phage typing of the staphylococci was not carried out, but a study of the sensitivity characteristics of the organisms in this case indicates that we were dealing with at least four different strains of the staphylococcus over the period of the patient's illness. (The apparent vagaries of sensitivity displayed by the organisms were probably due to sampling techniques.) At no time were the staphylococci sensitive to penicillin, and it is reasonable to suppose that the original pneumonia was due to the not uncommon "hospital" penicillin-resistant staphylococcus. There is no bacteriological support available for this assumption. The behaviour of the staphylococci here draws attention again to the versatility of the organism when faced with new therapeutic challenges, and to the disturbing epidemiological implications in man of its capacity to come to terms with a therapeutic environment.

The appearance of *C. albicans* in alarming proportions in the sputum during the course of antibiotic therapy exemplifies an aspect of biological behaviour which is becoming an increasingly important problem. This organism is ubiquitous, and it exists in a natural and delicate balance with the flora of the body surfaces including the mouth and the respiratory tract. It is becoming increasingly apparent that this balance can be disturbed by therapy, and in the treatment of an infection with an appropriate agent (controlled by sensitivity tests), a disturbance of the biological pattern may occur, which is characterized by an overgrowth of organisms unaffected by the particular agent. This may not be of serious moment; but in some instances, particularly when the therapy is continued for long periods, with a continuing upset of the pattern, the overgrowth can be of such intensity that serious if not fatal illness results. These so-called superinfections are most commonly the accompaniment of "broad spectrum" antibiotic therapy (chlortetracycline, oxytetracycline, tetracycline and chloramphenicol). Penicillin and the sulphonamides do not in general terms give rise to important complications of this sort. The possibility of superinfection was constantly before us during the course of our patient's illness, but it was considered justified to take the risk of such a development, in an attempt to control the staphylococcal infection.

C. albicans is a natural commensal, and the presence of Monilia in the sputum, even in association with antibiotic therapy, is no proof of pathological intent. Nor is response to therapy—for example, potassium iodide—necessarily significant in making a diagnosis of moniliasis. This diagnosis is hard to substantiate. Bronchoscopic evidence of a thrush-like bronchial ulceration, and necropsy evidence of monilial invasion of the interstitial tissue of the lung, make a diagnosis of pulmonary moniliasis virtually certain, although the latter evidence cannot be accepted unreservedly. Neither of these criteria was satisfied here, and it seems clear from this, and from the virtual disappearance of *C. albicans* from the sputum after cessation of the antibiotic therapy, that monilial infection did not play an important part in the course of the illness of our patient.

The ætiology of Sjögren's disease is obscure. Comment in the past has implicated many factors. Among these have been vitamin deficiencies, hormonal disturbances, infection (unspecified), and diseases of blood-forming organs. Without exception the evidence in favour of one or other of these factors has been conspicuously lacking in conviction. Ellman, Parkes Weber and Goodier have considered that the condition is due to an inflammatory atrophy of unknown origin affecting mucus-secreting and other glands. The resulting dryness of epithelial surfaces may give rise to obstructions in ducts and the bronchial tubes, with sequelæ familiar in these circumstances. The implication of an inflammatory atrophy is an unsatisfying conception; but the most we can offer in its stead is an equally nebulous suggestion that the disease results from the action of an unknown offending agent, having a predilection for epithelial tissues. In consequence atrophic changes occur in such tissues, and the cellular infiltration and fibrosis commonly seen in the tissue sections are secondary to the epithelial changes. There is an unusual similarity between the respiratory complications of this condition (seen here and in Ellman's case) and those of a disease essentially characterized by a disorder of mucus secretion —namely, mucosis. However, it is at this point that any real resemblance between the two conditions ceases.

Summary.

A fatal case of staphylococcal pulmonary infection occurring in a patient suffering from Sjögren's disease is reported. The initial illness complicated ACTH therapy. The ability of the staphylococcus to adapt itself to a therapeutic environment is illustrated, and the risk of superinfection attending the use of the "broad-spectrum" anti-

biotics is commented on. Superinfection played no part in the outcome of this case, but this complication may be, and often has been, of greater importance in influencing the outcome of an illness than any other factor.

The possibility of infection of the respiratory tract with drug-resistant organisms is a real one in these patients, and the risk becomes undeniable in circumstances such as accompany the use of ACTH and cortisone. Decisions to use these drugs in an attempt to ameliorate the symptoms of the disease or to control a polyarthritis which may accompany the state must be made with this contingency in mind.

Acknowledgements.

We are indebted to Professor E. S. J. King, Professor of Pathology, University of Melbourne, in whose department the sections were cut; to Dr. Michael Wilson, of the Public Health Laboratory, for help with the bacteriology; to Dr. L. C. Hudson, Medical Superintendent, Prince Henry's Hospital, for information about our patient from the hospital records; and to Dr. E. V. Keogh, Victorian State Director of Tuberculosis, for permission to publish this report.

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Reviews.

The Leaven of Love: A Development of the Psychoanalytic Theory and Technique of Sándor Ferenczi. By Izette de Forest; 1954. London: Victor Gollancz, Limited. 8″ × 5¼″, pp. 222. Price: 20s. 6d.

PSYCHOANALYSIS has given birth to an amazing literary progeny. Among the latest is one called "The Leaven of Love" which seeks to promulgate and advance the therapeutic ideas of Sándor Ferenczi, one of the earlier stalwarts who joined the Freudian hierarchy at the beginning of the present century and practised psychoanalysis until his death in 1933.

This book not only pays a warm tribute to Ferenczi in the short biographical account which occurs at the beginning, but in the later chapters it sets out his therapeutic ideas. Where Freud was passive, Ferenczi became an active psychoanalyst. He speeded up the analyses by active interference designed to help and to clarify situations as they arose. This method did not gain Freud's approval; but Ferenczi so loved and revered his master that he never sought to break with him, as Jung, Adler and Rank had done. And so sincere was Ferenczi that he not only gained many adherents, but achieved signal success with his neurotic patients. His was to a large extent a personal success; and the very elements which conspired to build this success are those which in other hands are liable to lead to weakness and emotionalism. What began as a science appears to have become a spiritual endeavour. So that the present author, beginning as a disciple of Ferenczi, is now become a Christian advocate, invoking the dual commandments of the Christian faith—the love of God and the love of one's neighbour—as the be-all and end-all of psychotherapy. Freud was once accused of developing a psychology without a psyche. Izette de Forest has sought to advance psychotherapy to a stage of spiritual healing based upon faith. The book's appeal will be chiefly to those ministers of religion, teachers and social workers who welcome an emotional approach to the subject.

Culture and Human Fertility: A Study of the Relation of Cultural Conditions to Fertility in Non-Industrial and Transitional Societies. By Frank Lorimer, with special contributions by Meyer Fortes, K. A. Busia, Audrey I. Richards, Priscilla Reining and Giorgio Mortara, and a foreword by Frank W. Notestein; 1954. Paris: United Nations Educational, Scientific and Cultural Organization. Melbourne: Oxford University Press. 9″ × 5¼″, pp. 514, with six text figures. Price: 31s. 3d.

THIS work purports to be a study of the relation of cultural conditions to fertility in non-industrial and transitional societies. Its first part is given over to problems of general theory adequately presented by Professor Lorimer; the remaining sections consist of a number of special studies undertaken by his collaborators, in which anthropological, demographic and social data are collected and appraised. Conclusions are often tentative, while some point to the necessity of additional research. Certain broad generalizations seem to admit of no doubt: the higher birthrate in the country as compared with the town, and the high level of fertility among men engaged in agriculture and allied pursuits as compared to that in urban pursuits. The strongest factor limiting birthrate in Brazil is the institution of marriage.

This is not a book for popular consumption. It is primarily for the demographer and the sociologist, and as a source-book for the anthropologist and the student of world affairs. A very large mass of material is sorted out; but some of the deductions are complicated and diffuse, and rather tantalizing to the untrained mind looking for broad generalizations and cogent definitions. It is believed by the Committee to which it owes its inception to be more useful in its present if somewhat unwieldy and tentative expression, than if it was more rigid and dogmatic, as it might have been if subjected to compromise and less original treatment. Eugenics exerts an increasing pertinence to the present-day world, and the study of population and fertility in different societies is demanding increasing attention. This book gives a worthy lead to an important investigation.

Treatment of Ear, Nose and Throat Conditions. A series of articles by various contributors, edited by W. Stirk Adams, M.B., Ch.B., F.R.C.S., and R. R. S. Strang, M.B., Ch.B., F.R.C.S., D.L.O., reprinted from the *Nursing Mirror*. 11¾″ × 8¼″, pp. 26, with 39 illustrations. Price: 2s. 6d.

THIS publication is sponsored by the Midland Institute of Otology. It consists of a series of articles presenting a comprehensive summary of many of the problems which may arise in everyday practice; they appear under such headings as "Foreign Bodies in Ear, Nose and Throat Practice", "Giddiness", "Nasal Catarrh", "Dysphagia", "Hoarseness and Stridor", "Pain and Headache in Otorhinolaryngology", "Deafness and Earache". In each section the basic plans of investigation and treatment are clearly explained and usefully illustrated with photographs, drawings and diagrams. The publication is reprinted from the *Nursing Mirror*, a fact which suggests a very useful purpose towards providing for the student nurse a helpful outline of the problems about which she must have a reasonable understanding. The hospital lecturer will find in these papers an excellent foundation for lectures to nurses and to medical students. Naturally there is more to be told than can be set out in twenty-four pages. The subjects which are covered, however, are clearly described in a well planned summary in each instance.

Cardiac Anomalies: A Clinicopathologic Correlation. By Vincent Moragues, M. D., and Chester P. Lynxwiler, M.D.; 1954. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 11¾″ × 9″, pp. 102, with 157 illustrations. Price: 70s.

ALL physicians who undertake the care of heart disease should possess an atlas of congenital heart disease, replete with reproductions of teleradiograms, electrocardiograms, angiocardiograms, and other relevant information, and with a minimum of letterpress. A recent attempt to provide this kind of assistance is this book, "Cardiac Anomalies", by Vincent Moragues and Chester Lynxwiler of St. Louis, United States of America. In their introduction they indicate three mechanisms by which the circulation adjusts itself to the altered morphology of congenital heart disease. Firstly, there may be complete pooling of venous and arterial blood; secondly, a unidirectional shunt may be established, and thirdly, multidirectional shunts may form. The presentation is divided into sections such as malposition of the heart,

defects of the cardiac septa, valvular anomalies, anomalies of septation of the *truncus arteriosus*, transposition complexes and anomalies of the aortic arch. The tetralogy of Fallot is described as a transposition complex, a rather doubtful classification. The letterpress is reduced to a few terse phrases, and while the reader is grateful for its brevity, it is sometimes inadequate or even erroneous. For instance, patients with auricular septal defect are said to show transitory cyanosis in infancy, early heart block, microcytic anæmia and increased right auricular pressure. We submit that this does not give the average clinical picture. Anomalous pulmonary venous drainage is wholly omitted. Similar half-truths appear elsewhere in this book. Observation of the jugular venous pulse is not mentioned. One could also gain the impression that an atrial septal defect almost always accompanies pulmonary stenosis, and that over-riding of the aorta is not the usual finding in pulmonary atresia with a ventricular septal defect rather than an interatrial communication or a patent *ductus arteriosus*. Pulmonary hypertension in patent *ductus arteriosus* is not mentioned.

The best features of this atlas are the quality of the publication and the numerous plates of pathological specimens and photomicrographs which reveal wide resources available to the authors. There are few references from text to plate to assist the student examining these illustrations, the electrocardiograms show standard limb leads only, and the Röntgenograms mainly exemplify advanced disease. A stranger to this field would have no appreciation of the relative incidence of the exempla. In our view, the atlas is best viewed as a specialized photographic museum, of chief interest to the pathologist rather than the clinician.

Bone: An Introduction to the Physiology of Skeletal Tissue. By Franklin C McLean, Ph.D., M.D., and Marshall R. Urist, M.D.; 1955. Chicago: The University of Chicago Press. 9″ × 6″, pp. 194, with 26 illustrations. Price: $6.00.

THE publishers state that this volume is the first of a series entitled "The Scientist's Library: Biology and Medicine". The purpose of the series is to acquaint the biologist with progress in specialties other than his own. This book fulfils this purpose admirably and the choice of the first title is a wise one. To the physiologist, bone is notoriously a subject upon which information is scattered and hard to find and a comprehensive survey is therefore of special value.

This book deals with every aspect of normal bone: histological, chemical, embryological and physiological. There is a chapter on healing of fractures and there are brief references to pathological topics of physiological interest, such as ectopic ossification, osteoporosis and rickets. The subject is dealt with from the beginning, but the possession of a biological background by the reader is assumed and the treatment is nowhere elementary. The style is generally clear and succinct; only in the chapter on the chemistry and crystal structure of the bone mineral does an excess of detail in too short a space cause some difficulty to the reader. It is interesting to note that the authors give cogent reasons for rejecting the view that the parathyroid hormone acts primarily on phosphate excretion by the kidneys in favour of the hypothesis of a direct action on bone.

This book may be confidently recommended to those who wish to bring their knowledge of the physiology of bone up to date.

Handbook of Radiology. Edited by Russell H. Morgan, M.D., and Kenneth E. Corrigan, Ph.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 7½″ × 5″, pp. 528, with 98 text figures. Price: $10.00.

THE editors of this work have chosen fifteen collaborators to produce a "compilation of quantitative data which, it is hoped, will be useful to workers engaged in the clinical, experimental and industrial applications of ionizing radiations".

Those who remember the United States of America Army X-Ray Manual of thirty-five years ago, edited by H. C. Ellsbury, or the recent "Physical Foundations of Radiology" (Glasser, Quimby, Taylor, Wetherwax), can take it that the size, the quality of binding, paper and illustrations are in the same first-class practical tradition. It is not surprising to find Glasser and Quimby among the collaborators.

The six major sections are described as: "Definitions of Physical Terms in Units", "General Physical Information (including Bio-physical Data)", "Radiotherapeutic Data (including Depth Dose Data for X Rays, Gamma Rays and High Energy Electrons, also Radio-Isotopes)", "Radiography and Fluoroscopy (including an illustrated section on Clinical Radiographic Technique)", "Radiation Protection".

An attempt to include in one handbook such a variety of work-a-day information is unlikely to have been made outside the United States of America. Even without the complication of catering for industry, the needs of the radiotherapist and of the radiologist no longer allow such a compact mutual satisfaction.

While one is uninterested in, say, the decay characteristics of frequently used radio isotopes or the calculated atomic attenuation coefficients of the elements for X and γ rays, the other is unlikely to require to know the trade names and special properties of water-soluble contrast media or the rating of particular brands of rotating anode tubes.

It is clear that in the United States of America the tradition of a general practitioner radiologist is dying hard.

On the other hand, this is a useful book for ready access in a large department, particularly in a hospital which has no medical physics section of its own.

For example, the radiotherapist or the senior radiotherapy technician will appreciate the comprehensiveness of the terms included in the sixty-nine pages of definitions and perhaps the usual "bible" assembled in such departments could be replaced by this collection of depth dose data and the rules and calculations in radium dosimetry.

Antenatal and Postnatal Care. By Francis J. Browne, M.D. (Aberd.), D.Sc., F.R.C.S. (Edin.), F.R.C.O.G., and J. C. McClure Browne, B.Sc., M.B., B.S., F.R.C.S. (Edin.), F.R.C.O.G.; Eighth Edition; 1955 London: J. and A. Churchill, Limited. 8½″ × 6″, pp. 680, with 94 illustrations. Price: 37s. 6d.

THIS book has for long been recognized as an excellent teaching and reference book on the subject. It now enters upon its eighth edition. For the first time there is a joint authorship, and the experience of the original author is augmented by the modern concepts of his son to produce the best edition to date. This is a happy collaboration, for the book has lost none of its unity of purpose.

The main complications of pregnancy and the puerperium are adequately dealt with in separate chapters, a method which makes it easy to refer to the correct place without delay. Perhaps the best portions of the book are those discussing diet in pregnancy (it has been shortened considerably), the toxæmias of late pregnancy and hæmorrhage in late pregnancy. The methods of preventing eclampsia by controlling the toxæmias are right up to the last minute of modern teaching, and a generous tribute is paid to the recent work done at the Women's Hospital, Crown Street, Sydney.

After a brief, interesting history of ante-natal care, the physiology and anatomy of pregnancy are dealt with, including the influence of the emotions. The correlation of the emotional and physical functions of the individual is handled by Dr. Grantly Dick Read and every practitioner would do well to read this and consider it, however much he may differ from some of its concepts. While it may be impracticable, or even undesirable in many cases, to have the husband present during the confinement, and while we may not agree that the child should be fed whenever it demands food and not by regulation, undoubtedly not enough attention is paid by the busy *accoucheur* to the mother's psychological make-up. It is interesting, too, to note that this programme of "painless" childbirth does not exclude the assistance obtained from an analgesic such as 100 milligrammes of pethedine.

Post-maturity is discussed in detail, including its medico-legal aspects, and the dangers of senility of the placenta and the fall of oxygen saturation of the fœtal blood are admitted, but advice is given against any interference. With this a number of obstetricians would disagree, and advocate induction of labour if post-maturity was a fully established fact.

Congenital malformations are discussed in much greater detail than is usual in such a manual as this, and the account includes Mendelism and sex linkage.

As stated already, those chapters dealing with *placenta prævia* and accidental hæmorrhage are particularly well done and offer useful summaries of treatment. The role of blood transfusion and the more frequent resort to Cæsarean section in *placenta prævia* are stressed as means of obtaining lower fœtal as well as maternal mortality figures.

Most obstetricians in this country would disagree with the author's treatment of contracted pelvis in a *primigravida* by the induction of premature labour by rupture of the membranes. They would hold that this method makes a trial labour, and possible Cæsarean section, more hazardous. Also they would not agree that it is possible for an *accoucheur* to estimate with any degree of accuracy the size of a fœtal head which will pass through the pelvis now but not in two

or three weeks' time. We must remember that the growth is only one millimetre per week. One is sorry to see the solid bougie (or the stomach tube) still advocated in these inductions.

Concomitant diseases of the circulatory, digestive, respiratory and urinary systems are adequately covered and practical advice is given on their modern treatment. With the termination of pregnancy at the thirty-seventh week in *diabetes mellitus* most of us will whole-heartedly agree, although many in this country would advocate artificial rupture of the membranes much more commonly than Cæsarean section.

An excellent chapter on radiology in obstetrics has been contributed by Professor Chassar Moir, of Oxford, and this, too, has been thoroughly revised and brought up to date. It includes methods of estimating pelvic diameters and the most recent work on placentography.

One of the main difficulties encountered when producing such a comprehensive work as this is to limit the volume to a size which can be handled with comfort. This has been accomplished and the book can be confidently recommended to all practitioners who conduct midwifery cases, and more especially to post-graduates studying for the M.R.C.O.G. diploma.

Genetics and the Inheritance of Integrated Neurological and Psychiatric Patterns: Proceedings of the Association for Research in Nervous and Mental Disease, December 11 and 12, 1953. Edited by Davenport Hooker, Ph.D., and Clarence C. Hare, M.D.; 1954. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. Volume XXX. 9" x 6¼", pp. 434, with 77 illustrations. Price: £5 7s. 6d.

IN 1923 there appeared Volume III of the Research Publications of the Association for Research in Nervous and Mental Disease, devoted to the subject of inherited factors. Now, after thirty years, Volume XXXIII has been issued with the lengthy title "Genetics and the Inheritance of Integrated Neurological and Psychiatric Patterns". In this long interval of time—long in modern biological and medical history—the science of genetics has made enormous strides and the direct applications to practical medicine are increasing at a rapid rate. Thirty authors are responsible for twenty-four articles which, the reader is informed, deal only with a segment of the whole subject; but this segment is assuredly of major importance. Whether the editor, Dr. Davenport Hooker, Professor of Anatomy in Pittsburg, merely chose his team wisely or exercised throughout a critical supervision or combined both of these, we are not told, but a scientific spirit of high order pervades all the contributions; there is a commendable caution and an absence of catchy surmises. The first chapter, by Paul R. David and Laurence H. Snyder, gives a masterly summary of the principles of human genetics as understood today. The exposition displays a minimum of the formidable terminology which too often frightens away prospective readers not well versed in genetical theory. One learns how complex the subject is and how difficult it is to disentangle innate from acquired characters. For instance, there is a familial type of cretinism, indistinguishable from nutritional cretinism but occurring in districts well supplied with iodine. Again, the environment can modify genotype departures from normal and disguise or intensify these. One realizes on perusal of this book how important "identical twins" are in human genetic investigations, especially if the twins are brought up in diverse environments. We must point out that such twins are never quite "identical"; the fission of the ovum may be morphologically perfect, but the probabilities of exactly equal placental nutrition are remote; further, the intrauterine packing shows itself in certain physical features such as the variability in the convexity of the forehead.

Amongst articles of high level of excellence it may seem invidious to single out any one for special comment, but the chapter entitled "The Hereditary Ataxias" by John W. Schut is worthy of favourable mention. An investigation on the fifth cranial nerve may be thought to belong more to embryology than to genetics, but the inclusion can be justified; in the same connexion there is a survey of the results of surgical tractotomy (a sad hybrid) with regard to trigeminal pain. Altogether the book must be commended, not only for the scholarship, but for the sound common sense presented throughout. Impetuous advocacy of certain eugenic measures will be inhibited. In a very full discussion on epilepsy in its genetic and eugenic aspects there occurs the (to some) startling conclusion: "We believe that most epileptics who wish to marry and rear children should be encouraged to do so." We trust that a supplementary volume will appear in much less than the thirty years separating this from its predecessor.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Clinical Pathology in General Practice: Specially Commissioned Articles from the British Medical Journal, October, 1953, to July, 1954"; 1955. London: British Medical Association. 8½" x 5½", pp. 332, with many illustrations. Price: 21s.

Articles reprinted from the *British Medical Journal* designed to inform the general practitioner of the kind of help the clinical pathologist can provide him and to give him some guidance in the interpretation of pathological reports.

"Lectures on the Scientific Basis of Medicine: Volume III, 1953-1954"; 1955. London: University of London, The Athlone Press. 8½" x 5½", pp. 416, with nine illustrations. Price: 35s.

This book bears the imprint of the British Postgraduate Federation; there are 21 chapters.

"Mitchell's Anatomy and Physiology, and Causes of Disease", by H. C. Trowell, O.B.E., M.D., F.R.C.P.; 1955. London: Baillière, Tindall and Cox. 6¼" x 4", pp. 260, with 87 illustrations. Price: 7s. 6d.

Designed for students in tropical and subtropical countries whose knowledge of English is too limited to enable them to use standard English text-books.

"The Management of Obstetric Difficulties", by Paul Titus; Fifth Edition; revised by J. Robert Willson, M.D., M.S.; 1955. St. Louis: The C. V. Mosby Company. Melbourne: W. Ramsay (Surgical), Limited. 9" x 6¼", pp. 738, with 348 illustrations. Price: £6 17s. 6d.

Though the scope of the book has been expanded its purpose is still to provide a practical aid to obstetricians.

"Fluoroscopy in Diagnostic Roentgenology", by Otto Deutschberger, M.D., with an Introduction by Frank J. Borrelli, M.D., F.A.C.R.; 1955. Philadelphia and London: W. B. Saunders, Company. Melbourne: W. Ramsay (Surgical), Limited. 10" x 7", pp. 790, with 523 illustrations. Price: £11.

Intended to meet the deficiency of literature on fluoroscopy.

"*Neuropharmacology: Transactions of the First Conference, May 26, 27 and 28, 1954, Princeton, N.J.*", edited by Harold A. Abramson, M.D.; 1955. New York: Josiah Macy Junior Foundation. 9" x 6", pp. 210, with 44 illustrations. Price: $4.25.

The subjects discussed at the conference were: "Considerations of the Effects of Pharmacological Agents on the *Over-All Circulation and Metabolism of the Brain*", "Functional Organization of the Brain", "Studies of Electrical Activity of the Brain in Relation to Anæsthesia", "Ascending Reticular System and *Anæsthesia*", "*Observations of New* CNS Convulsants".

"*Blutgerinnungsfaktoren*", by Erwin Deutsch; 1955. Vienna: Franz Deuticke. 9½" x 6½", pp. 312, with many illustrations. Price: DM42.

The book is divided into sections dealing with the successive phases of blood coagulation.

"The Year Book of Endocrinology (1954-1955 Year Book Series)", edited by Gilbert S. Gordan, M.D., Ph.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 8" x 5", pp. 392, with 94 illustrations. Price: $6.00.

One of the Practical Medicine Series of Year Books.

"The Year Book of Dermatology and Syphilology (1954-1955 Year Book Series)", edited by Marion B. Sulzberger, M.D., and Rudolf L. Baer, M.D.; 1955. Chicago: The Year Book Publishers, Incorporated. 8" x 5", pp. 472, with 59 illustrations. Price: $6.00.

One of the Practical Medicine Series of Year Books.

"The Surgical Clinics of North America"; 1955. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. New York Number. 9" x 6", pp. 308, with 151 illustrations. Price: £8 2s. 6d. *per annum* in cloth binding and £6 15s. *per annum* in paper binding.

This New York number consists of a foreword and twenty-one articles dealing with critical emergencies. There are 35 contributors.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 3, 1955.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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THWARTING THE AUTOMATON.

NATURE in her infinite wisdom—the pre-Darwinian phrasing remains by reason of its ease of expression—has ordained that voluntary control should be confined to skeletal muscle, and hence the working of smooth muscle, heart muscle, glands and the mysterious happenings in growth and embryological development should be detached from volitional interference. Even voluntary muscle is not always so completely guided by the will as is imagined. Buckle aroused the anger of mid-Victorian fundamentalists by declaring that the number of suicides and of marriages in a sufficiently large community varied with the quality of the season. Modern physics has shown that though the molecule flits to and fro in an unpredictable path the mass movement of a large aggregate of molecules, sufficiently numerous to allow statistical methods to be applied, follows fixed laws, and so accurate calculation is possible. What appears in the individual human being to be purely voluntary and the product of free will follows definite trends in communities. In the case of the regulation of activities by autonomic nerves and by hormones, deliberate and conscious guidance is impossible except in a few instances by very devious routes. Let us take eating as an example. The choice of foodstuffs seems to be voluntary, yet Irving Fisher in 1906 showed that the normal human being, completely unaware of what he is doing, selects combinations of foodstuffs which in the aggregate display definite ratios between proteins, carbohydrates and fats. It is true that in cold weather there is a shift towards the fatty side and a release from this on the return of warmth, but the fact remains that the distribution of the three main sources of chemical energy in a meal is strikingly uniform and is all the more remarkable in that the selection takes place unconsciously. When food is admitted to the mouth mastication is a voluntary effort, but the flow of saliva which shows some adaptation to the food, not as intimate perhaps as Pavlov believed, is an involuntary reflex. Once the food bolus has passed the fauces and entered the œsophagus all voluntary direction and supervision cease. The activities, physical and chemical, of the stomach, by no means yet all revealed, the digestive and absorptive operations of the intestines, the manifold functions of the liver, all take place secretly and unknown to us except when something goes wrong. That there must be a purpose (again pre-Darwinian wording is chosen) for so many activities of the body to be sealed and sequestered from volitional interruption must be obvious. Possibly, some may say, inexorable natural selection destroyed those mutations which permitted what in the others, the survivors, was forbidden.

The arrival of *Homo sapiens* on the evolutionary scene witnessed the beginning of a mighty change in the automaticity of the body, for it was found that certain chemical substances which could be deliberately prepared or culled could operate on tissues activated normally by hormones or autonomic nerve reflexes. It is true that certain insects like the bees can permit or prohibit the development of sexual maturity in the female by dietary regulation, but it is in man that the impingement of volition upon the hitherto sheltered and involuntary activities of the body is most pronounced. Fermentation of sugary fluids produced ethyl alcohol, perhaps the earliest of such agents, and much ingenuity has been exercised on the preparation of fermentable liquor from starch and also on the selection of organisms which operate on milk sugar. The pharmacology of alcohol presents us with an enormous volume of research dealing with the detailed reactions on various tissues and organs, but primarily the basic property which makes alcohol popular is the power it gives to change at will what without it would remain immune to interference. Oliver Wendell Holmes in his "Autocrat of the Breakfast Table" has referred to this compelling desire to break into automaticity:

> Will nobody block those wheels, uncouple that pinion, cut the string that holds those weights? . . . If anybody would only contrive some kind of a lever that one could thrust in among the works of this horrid automaton and check them or alter their rate of going what would the world give for the discovery? From half a dime to a dime according to the style of the place and the quality of the liquor, said the young fellow whom they call John.

It may be mentioned here that Holmes attacked the temperance propaganda of his day, nearly one hundred years ago, with vigour. They pulled out the tail of the burrowing Guinea worm but left the head, he declared. Had his wise admonitions been remembered and studied, the unfortunate prohibition laws of the United States would never have been enacted. Alcohol, opium, Indian hemp and even poisonous fungi have been used to break into the automatic security of the viscera, often "brutalizing the machine". Tobacco smoking may be regarded as a mild form of drug addiction, but its widespread popularity calls for explanation. As one pharmacologist wittily remarked: "It is a poison all right, but it takes 80 years to kill." Today we are entering on a new era of deliberate regulation or at least alteration of hidden body functions. Hormones are prepared synthetically and placed on the market by commercial chemists, and there seems to be no

limit to what may be attained by further research. Application of sex hormones may effect profound alterations in our social life; for example, a contraceptive drug could bring about serious disturbances in what has hitherto been regarded as a moral sphere of action. Male and female attributes and appearances may be interchanged—in a "Current Comment" in this journal a description was given of a normal boy of fifteen years who worked in a factory making ovarian products and inhaled sufficient hormone for him to develop female mammæ and to be threatened with atrophy of his male genitalia. The medical profession must be prepared to watch such possibilities and with wisdom to advise legislators. The atom bomb may jeopardize the continuance of the human species; voluntary regulation of the body's functions, hitherto beyond our powers, may induce momentous changes in our civil and domestic life which cannot now be predicted and which must be met with calmness and sagacity as they arise.

Current Comment.

CONVULSIVE TREATMENT FOR PSYCHIATRIC ILLNESS.

AN interesting contribution to the study of convulsive treatment for psychiatric illness has been made recently by R. Redhardt.[1] The paper, which covers ten years' experience with convulsive treatment, differs from the usual statistical reports in its attempt at a highly specific diagnosis and in the very careful indications it lays down for convulsive treatment. Thus, although the percentage of psychiatric patients treated by Redhardt increased over the years on the basis of previous experience, only 10% of the 8900 patients of the series were treated with electroconvulsive therapy or "Cardiazol" shock. Redhardt emphasizes the view that neurotics and psychopaths should be given shock treatment only if symptoms constitute a danger to life; of over 5500 cases in his series shock treatment was given in only 0·8%, but with good results.

The main groups from which patients were selected for shock treatment were schizophrenics (1307 subjects) and those with manic psychoses (2129 subjects), of which 23·7% and 25·6% respectively were given shock treatment. Only rarely, on vital indication, was shock treatment given on consecutive days; the routine was two shocks per week to a total of 15 or 20 shocks. In 10% to 11% of the patients treated anxiety reactions were observed. Transient amnestic disturbances appeared after the majority of electric shocks, but considerably less often after "Cardiazol" shock. Redhardt does not seem to consider these functional disturbances as a contributing factor to therapy, since the treatment was interrupted if very pronounced symptoms occurred. In cases of schizophrenic psychoses "Cardiazol" shock was found to be distinctly superior to electric shock (25·4% compared with 15·4% of good results). In most subgroups of the disease the effect in relation to cure or improvement was found to be discouraging or nil. Distinct benefit was obtained only in cases of confused schizophrenia, of atypical catatonia (iterative—excited catatonia) and of atypical paranoia (progressive reference psychosis). Patients with typical paranoia and hebephrenia showed almost no response; those with typical catatonia, though not showing evidence of cure, responded with about 20% of good and 30% of slight improvement. However, it must be mentioned that the criteria for cure used by Redhardt are very strict—namely, that careful expert investigation can detect no more than slight logic defects, or narrowing of affective width, or loss of mimic and other expression; furthermore the patients are classified as not having received benefit if only temporary relief from one or other prominent symptom was achieved.

With regard to the phasic psychoses, no difference was observed between the effect of "Cardiazol" and that of electric shock, and the therapeutic result was uniformly good, being better in the monophasic depressive forms than in the monophasic manic forms (67% and 34·7% of cure respectively). The result was intermediate in the cyclic forms (55·8% cure); here again the depressive phase responded better than manic conditions. The therapeutic effect was the same in first and in later attacks of the illness, but the incidence of recurrences was not affected.

The importance of detailed diagnostic differentiation, particularly of the schizophrenias, is stressed by Redhardt in his conclusion. He points out that in the last-mentioned conditions steadily progressive typical forms as well as disturbances of affect were not influenced, and psychomotor disturbances, excitement or the affect-content of hallucinations and paranoid complexes were only temporarily reduced.

[1] *Arch. Psychiat* (1954), 192: 328-355.

A NEW TREATMENT FOR SEBORRHŒIC DERMATITIS.

A NEW group of organic fungicides was introduced in 1952 by Kittleson. One of these, known as "Captan" or "Vancide 89", with the formula N-trichloromethylmercapto-4-cyclohexane-1,2-dicarboximide, has been successfully used as an agricultural fungicide; in purified form it is colourless, odourless and water-soluble. Franklin I. Ball,[1] searching for a more effective treatment for seborrhœic dermatitis, made trial of this drug. There was evidence of its low toxicity from the tests made before it was used in agriculture. After purification by recrystallization, a lotion was made up of "Vancide 89", 0·085%, and benzethonium chloride ("Phemerol"), 0·060%, in water and alcohol; this was labelled "Solution 105-6", and is now being marketed as "SEBB". It is supplied in a shaker-top bottle, and is sprinkled onto the scalp and massaged lightly in once a day.

Ball tested this treatment on 90 patients with seborrhœic dermatitis and eight with psoriasis of the scalp; their ages varied from three months to eighty-two years. The psoriasis patients showed no improvement; only four of the others showed no improvement at all, and of eight who had *otitis externa* four showed no improvement. Associated acne in adolescents was also benefited. Patch tests produced no irritation or sensitization, but two of the patients suffered reactions, described as "allergic", which prevented continuation of treatment.

In the same journal, Stephen T. Whelan reports on the use of a cream-type, greaseless lotion of 10% sodium sulphacetamide ("Sebizon Lotion") for seborrhœic dermatitis; it was massaged into affected sites twice a day. Of 61 patients with scalp lesions, 54 showed good improvement, while of 44 with the glabrous skin involved, only 30 improved. In seven patients the lotion caused reactions severe enough to necessitate stopping the treatment, but there was no true contact dermatitis.

Possibly these are only two additions to the already long list of more or less effective treatments for seborrhœic dermatitis, but they both seem as if they might be worth a trial.

[1] *Arch. Dermat.*, June, 1955.

PENTAERYTHRITOL TETRANITRATE ("PERITRATE").

THE therapeutic evaluation of new drugs is often difficult, and this is particularly so when most of the indications for the effects of the drug are subjective. *Angina pectoris* is a condition in which the chief, if not the only, symptom

is pain in the chest which may or may not radiate to the neck and left arm. Changes in the electrocardiogram have been noted, but according to some observers are not always present during an attack. Some authors believe that true *angina pectoris* is always relieved by trinitrin and that this is a diagnostic point. Many drugs have been tried in the search for one which will act much longer than trinitrin and varying degrees of success have been claimed for several of them. One of the newest is pentaerythritol tetranitrate ("Peritrate"), and this drug demonstrates very well the difficulty of therapeutic evaluation. The manufacturers of "Peritrate" claim that it prevents attacks of *angina pectoris* in four out of five cases and that prophylaxis with "Peritrate" "results in fewer, less severe attacks, reduced nitroglycerin dependence, improved E.K.G.'s where abnormal patterns exist and increased exercise tolerance".

The first detailed account of its pharmacology was made by von Oettingen and co-workers in 1944.[1] Not much was done about it until 1952 when Winsor and Humphreys studied the effect of pentaerythritol tetranitrate on 125 patients with *angina pectoris*. It was claimed that an average dose of 10 milligrammes three times a day produced satisfactory prevention of precordial pain in 78·4% of the patients and that the trinitrin requirement was reduced, and exercise tolerance increased. M. Plotz[2] in 1952 made similar claims in a study of 35 cases. H. I. Russek[3] and co-workers in 1953 compared nitroglycerin and pentaerythritol tetranitrate with a number of other drugs which had been recommended. Of all the agents studied pentaerythritol tetranitrate appeared to be the most effective compound in producing prolonged prophylactic therapy in *angina pectoris*. In a further study in 1954 H. I. Russek[4] and co-workers examined a large number of drugs by comparing their ability to modify the electrocardiographic response to standard exercise in patients with *angina pectoris*. "Peritrate" was claimed to be the most effective for prolonged prophylactic effect in *angina pectoris*. Recently E. W. Snow[5] claimed that pentaerythritol tetranitrate with "Rauwiloid" gave a great increase in exercise tolerance in patients with *angina pectoris*. Other apparently equally good and careful observers have obtained very different results. D. Weitzman[6] gave 34 patients pentaerythritol tetranitrate and a similar 31 patients a placebo. The first group showed greater benefit than the first, but this did not hold when the groups were reversed. A. H. Salans and co-workers[7] in a careful reappraisal found that pentaerythritol tetranitrate produced no significant benefit in patients with *angina pectoris*. J. E. F. Riseman and co-workers[8] in 1954, in a very careful study of the effects of cinchona alkaloids and a number of other drugs in *angina pectoris*, gave 50 milligrammes of pentaerythritol tetranitrate four times a day, a very much larger dose than is usually given, and found a favourable response in only three of fifteen patients.

The latest report is by G. M. Kalmanson and co-workers.[9] They studied the effect of pentaerythritol tetranitrate on *angina pectoris* in 23 patients by alternating placebo medication with the drug, using the very efficient "double blind" method. No beneficial effects of the drug were observed. A careful study of the papers mentioned leads one to believe that there was nothing to choose between the various groups of workers in care of observation and detailing of experiments. The "double blind" method of noting the results may give somewhat more accurate results than the methods used by other observers. In any method used the patient has to compare intensity of pain with that experienced on some prior occasion, and this is obviously extremely difficult. The determination of the number of attacks in a day is not reliable when the patient has to note them. Further, so many factors can influence the number and intensity of anginal attacks that observations would have to be made over very long periods to allow for their influence.

No one can say at present whether pentaerythritol is of any use in the prophylactic therapy of *angina pectoris*, and the personal opinion of any practitioner without detailed observations of patients has no value.

[1] *Angiology*, February, 1952.
[2] *New York State J. Med.*, August, 1952.
[3] *J.A.M.A.*, September 19, 1953.
[4] *Am. J. Med. Sc.*, January, 1955.
[5] *Northwest Med.*, January, 1955.
[6] *Brit. Med. J.*, September 19, 1953.
[7] *J.A.M.A.*, November 21, 1953.
[8] *Circulation*, December, 1954.
[9] *Arch. Int. Med.*, June, 1955.

DEPENDENCE ON AN "IRON LUNG".

As with other muscle groups, there is usually a considerable amount of recovery from the original extent of poliomyelitis respiratory muscle paralysis, but until the introduction of tank respirators in about 1930 there was no way of keeping a severely paralysed patient alive for long enough to enable recovery to come about. Even so, before the days of antibiotics those patients who were tided over, if they had much residual paralysis, died fairly soon from pneumonia. But nowadays there are increasing numbers of patients who have been left with a degree of respiratory paralysis which makes them entirely dependent on an "iron lung" for life. However, J. C. Wilson and D. G. Dickinson,[1] discussing this problem, state that the number of patients confined to these machines is far greater than it need be. It is their opinion that with skilful and energetic treatment, carried out decisively toward a single purpose, it is almost never necessary for a patient to be confined in such a device indefinitely. The limiting factor is the number of anterior horn cells which remain able to function, but the factor chiefly responsible for keeping patients in respirators unnecessarily is psychological; there is a physical factor involved, too, which has been named "pulmonary compliance".

If we concede that any patient who can remain alive with the assistance of a tank respirator must have some residual function left in his respiratory muscles, the true extent of his independence can be discovered only by determined efforts. If his pulmonary compliance is good enough—that is, if there is not too great a loss of the elasticity and expansibility of his lungs and thoracic cage, due to muscle spasm, to the development of contractures of connective tissue and fixation of the muscles and joints of the thorax, and to cohesion of the moist respiratory surfaces which have not been frequently and fully expanded—these efforts may be made. If his pulmonary compliance is poor, it may be improved by heat, massage and muscle stretching, by periodic overstretching of the lung with deeper negative pressures obtained by rhythmically introducing the hose of a vacuum cleaner through one of the ports of the respirator, by reversing the vacuum cleaner hose to the exhaust side and attaching it to a face mask for positive pressure breathing, and by "frog-breathing", which consists of blowing up the lungs by rapidly swallowing air into the trachea.

Many patients lack incentive to try to dispense with their respirators. When these machines were first introduced, poliomyelitis was almost entirely confined to young children, who, as soon as they were well enough, were eager to get out, and who were more amenable to the disciplines necessary to train them to this end. Nowadays the majority of respirator patients are older. They can realize more fully the implications of their disability; they can visualize the unending struggle they must face to achieve even a partly normal life, and can estimate their chances of earning a living. To many their respirator life must seem their only security; others will have given up hope entirely, relapsing into an infantile or a vegetable existence. Since their determined cooperation will be needed to get them out of their respirators, such patients present difficult problems.

Wilson and Dickinson then discuss what they call "psychogenic hyperpnœa". They state that early in the

[1] *J.A.M.A.*, June 18, 1955.

course of poliomyelitic respiratory paralysis many patients develop a hyperpnœa, with a strong demand for excessive pulmonary ventilation; most of them ask for more ventilation in the respirator than they really need, because they have a very real, even if only psychogenic, respiratory distress. The consequence is that many patients are sustained in a state of respiratory alkalosis, which becomes their only state of comfort. Wilson and Dickinson have decided, after considering all the evidence at their disposal, that this state is not the result of any disturbance in respiratory physiology, but is due to subconscious or conscious fear of respiratory failure, and they describe it as a form of "addiction". At any rate, they have found it necessary to estimate this hyperpnœa in most patients and correct it. They measure vital capacity and tidal air, and calculate alveolar carbon dioxide tension; from these data they work out how much to reduce hyperventilation, and then they wean the patient from his respiratory alkalosis. Thereafter, by increasing periods of reduced respirator excursions, then unassisted breathing, the patient is trained to become independent. Some achieve only short periods of freedom, some need to return to the respirator for sleeping, but many can get along with a cuirass respirator by day and a rocking bed at night, and some even become independent of any mechanical aids.

Wilson and Dickinson have had remarkable success with their methods, and their paper offers real encouragement to all those who may have the care of patients with respiratory paralysis.

CARDIOSPASM.

GEOFFREY H. WOOLER[1] has pointed out that the number of synonyms found in the literature for the condition usually called cardiospasm (achalasia, œsophagectasia, idiopathic dilatation, mega-œsophagus, neuropathic dilatation, phrenospasm) is in itself an indication of the wide diversity of opinion regarding the cause of this disease. Wooler accepts the view that probably several factors contribute to the onset, but he regards psychogenic trauma as by far the most important. (He is, it seems relevant to note, a thoracic surgeon.) At the same time he states that in his experience the lesion appears to be organic and is not cured by psychotherapy; and when he goes on to discuss treatment, he includes psychiatric advice in medical treatment, but discusses methods of dilatation and operative measures at length.

It must be conceded that in a condition such as cardiospasm the role of psychogenic factors is difficult to assess. In a recent review of the subject P. H. Holinger, K. C. Johnston and M. F. McInnes[2] refer to the fact that the role of such factors has been stressed by some and denied by others. In their own experience, they have found evidence of obvious psychogenic stress in 17 of a series of 189 cases. Their comment is that where a correlation does exist, the question may be raised whether the psychogenic symptoms are the cause or the result of the chronic swallowing difficulty. And that seems to be about as far as the matter has gone. The available evidence, according to these investigators, supports the view that the neuromuscular function of the entire smooth muscle of the œsophagus is at fault. Years ago Mosher stated that spasm played but a minor part in the cases that he met with in hospital; and it has been demonstrated that there occur loss of tonus, lack of propulsion and an irregular disorganized wave pattern in the upper part of the œsophagus of persons suffering from cardiospasm. The extreme degrees of dilatation seen in association with cardiospasm are rarely seen in association with other fibrotic and neoplastic lesions occurring at the cardia. The weight of evidence apparently favours the view that cardiospasm is primarily a disease of the myenteric plexus.

Holinger, Johnston and McInnes review some of the clinical features demonstrated in their series of 189 cases, occurring in patients aged from six to eighty-three years and with an approximately equal sex distribution. It is instructive to consider the main points brought out. Holinger, Johnston and McInnes state that the most common and distressing symptom is dysphagia. This is usually intermittent at first, although occasionally the onset is abrupt and rapidly progressive. A variety of sensations result from the delay in œsophageal emptying. These may be described as feelings of retrosternal or epigastric fullness, pressure, heaviness or vague discomfort. Severe pain usually signifies an associated œsophagitis. Weight loss may be considerable and is associated with weakness simulating the cachexia of malignant disease. Symptoms of avitaminosis may occur with the general nutritional deficiency. In occasional cases, however, patients are actually obese in spite of a high degree of stenosis. The symptoms of œsophageal overflow may be particularly distressing, since the patient may be unable to eat in public on account of vomiting or regurgitation. Often when he is in the recumbent position at night, the pharynx is flooded with the contents of the evening meal. Chronic aspiration of œsophageal contents leads to a variety of pulmonary complications, even sudden death, as occurred in one eighteen-year-old boy of the series under review.

The differential diagnosis of organic from functional obstruction is of utmost importance. Holinger, Johnston and McInnes point out that the symptoms of cardiospasm may fluctuate for a considerable time, often influenced by emotional tension, whereas the symptoms of a patient with carcinoma progress rather steadily. The history is often a long one in cardiospasm; whereas that of a patient with carcinoma is shorter. X-ray studies establish the diagnosis in the greatest number of cases. Cardiac enlargements, mediastinal lesions, pulsion diverticulum, peptic ulcer with fibrosis, congenital short œsophagus, hiatal hernia and epiphrenic diverticulum must be considered. Œsophagoscopic studies are considered a routine and essential step in establishing the diagnosis, as well as in controlling therapy.

The value of drug therapy in early cardiospasm is unquestioned. Symptomatic relief is obtained through the use of belladonna derivatives and nitroglycerin. The emptying of the œsophagus under the influence of these agents can be observed under the fluoroscope. However, they are of little or no value in far advanced cases. A thorough investigation of the remainder of the gastrointestinal tract and the gall-bladder should be made. Cardiospasm may be associated with upper abdominal disease and may give way to proper management of the latter. In this series eight peptic ulcers were found, one œsophageal, two gastric and five duodenal. There was evidence of gall-bladder disease in nine cases.

Active therapy consists of intraœsophageal dilatation and the external surgical procedures. The presence of ulceration may call for appropriate dietary and medicinal treatment first. Dilatation under fluoroscopic control is a more certain procedure than blind bouginage. Under fluoroscopic observation dilating instruments may be seen merely to turn back on themselves, failing to enter the actual constriction. Best results are obtained if the pneumatic or hydrostatic bag is placed in the stricture over a string guide, and the bag inflated several times to the point of slight epigastric distress. Actually such a treatment amounts to an intraœsophageal muscle-splitting procedure. To dilate beyond the limits of mucosal elasticity will result in a mucosal tear. Two perforations occurred in the series while the patients were undergoing pneumatic bag dilatation. Both patients survived, one after surgical repair and the other after conservative therapy. The Heller muscle-splitting operation seems best suited for cases of tubular œsophageal dilatation without evidence of a severe fibrous stricture. More extensive surgical procedures may be required. Resection with gastro-œsophageal anastomosis results in the greatest number of failures owing to regurgitation of stomach contents, œsophagitis and stricture formation. Surgery directed toward alleviation of the obstruction is inadequate unless it includes a procedure designed to reduce the acid secretion of the

[1] "Modern Trends in Gastro-Enterology", edited by F. Avery Jones, Butterworth, 1952.

[2] *Ann. Otol., Rhin. & Laryng.*, March, 1955.

stomach. This may be done by partial resection or by vagotomy.

The experience of Holinger, Johnston and McInnes has been that dramatic immediate relief is obtained from almost any kind of dilatation or surgical correction. Nevertheless, recurrence rates are high after either type of management, and it cannot be said that the treatment of cardiospasm is yet wholly satisfactory.

CHLORPROMAZINE AND VOMITING.

OVER the past two years a number of reports have been published on the value of chlorpromazine for the control of nausea and vomiting. D. G. Friend and J. F. Cummins[1] obtained gratifying results in a series of 70 patients who either were suffering from various major pathological conditions that produce vomiting or were experiencing vomiting induced by drugs. The conditions included uræmia, labyrinthitis, carcinomatosis, lymphomatosis and vomiting of pregnancy. The drugs included "Antabuse", folic acid antagonist, chlortetracycline ("Aureomycin"), codeine, meperidine, methadon, morphine, nitrogen mustard, oxytetracycline ("Terramycin"), protoveratrine and methane. No serious side-effects were noted.

Others have reported similar results, including relief from nausea induced by X-ray therapy, and now C. W. Daeschner *et alii*[2] have described their experience with chlorpromazine in the control of vomiting from a wide variety of causes in children. Such variable conditions as gastro-enteritis, diabetic ketosis, irradiation sickness, functional vomiting and bulbar poliomyelitis are included in the study. In 100 trials the drug gave excellent symptomatic relief of the vomiting in over 80% of cases; in only 8% was there no improvement. Daeschner and his colleagues observed that there was often a mild but useful soporific effect in addition to the antiemetic one, and that if chlorpromazine was combined with a sedative it seemed to potentiate the latter. They give warning (it should scarcely be necessary) that the cause of the vomiting should always be determined before the drug is given, for otherwise significant and dangerous symptoms may be masked. Thus its use as an antiemetic in cases of intestinal obstruction or central nervous system injury is debatable. In patients receiving the drug for more than a few days the possibility of agranulocytosis arises. It is probably unwise to give the drug to patients suffering from jaundice or liver disease, and certainly its administration should be stopped if a patient develops jaundice while taking it.

The effective dose of chlorpromazine varies with the route of administration. Daeschner *et alii* found that a dose of 0·5 milligramme per kilogram of body weight was usually effective by intramuscular injection, while twice that dose was required if given by mouth. Tablet and syrup preparations seemed equally effective by mouth. Given as rectal suppositories, a dose of two to three milligrammes per kilogram of body weight was required to obtain a satisfactory result. None of these dosage forms produced any evidence of local irritation.

ANTIHISTAMINIC DRUGS AND TRANSFUSION REACTIONS.

ALTHOUGH the technique of blood transfusion has been developed to a remarkable standard of safety, there are still certain hazards which may occur from time to time. Of these, hæmolytic reactions due to the administration of incompatible blood are the result of faulty technique or mere human carelessness; homologous serum jaundice can steal past the most careful screening of donors, and circulatory overloading is a purely mechanical effect, due to giving too much blood too fast. But allergic and anaphylactic reactions, and pyrogenic reactions, are due to inherent defects in the donated blood itself, and C. R. Stephen, Ruth C. Martin and M. Bourgeois-Gavardin[1] have investigated the effect of antihistaminic drugs in preventing these conditions. For twelve months all adult patients who received transfusions in Duke Hospital were followed carefully. During the middle part of this period 1616 patients were given tripelennamine, 25 milligrammes of which were added to the first 500 cubic centimetres of blood just before transfusion commenced. During the first and last parts of the year 1515 patients were given transfusions without any tripelennamine. Of the untreated patients 5·8% suffered pyrogenic reactions and 3·1% suffered allergic reactions, while for those treated, the figures were 3·4% and 0·6%; these figures are statistically significant. In the same issue of the journal, R. E. Wilhelm, Helen M. Nutting, H. B. Devlin, E. R. Jennings and O. A. Brines have published a report concerning the effect of diphenhydramine hydrochloride on allergic and pyrogenic transfusion reactions. After having shown that diphenhydramine in a concentration of 0·01%, when added to stored whole blood, did the blood no harm, a series of bottles was prepared and given as a routine to unselected patients. Of 495 treated patients, 2·2% suffered pyrogenic reactions and none suffered allergic reactions; among 453 control patients, the figures were 2·0% and 1·1%. These workers conclude that diphenhydramine hydrochloride will eliminate allergic reactions, but has no effect on the incidence of pyrogenic reactions. They also carried out experiments on rabbits, in which a known pyrogenic material derived from a culture of *Pseudomonas æruginosa* was injected; they found that diphenhydramine, chlorprophenpyridamine and tripelennamine all prevented pyrogenic reactions.

The occurrence of allergic reactions after blood transfusions has been reported as 2·2%, and of pyrogenic reactions as 2·6%, by the Mayo Clinic. Even though few pyrogenic reactions are dangerous, it would be a very good thing to have some sure means of eliminating them, along with allergic reactions. It would seem that although opinions vary on the efficacy of antihistaminics in preventing pyrogenic reactions, some are of value for preventing allergic reactions, although the perfect drug has yet to be found. The search will no doubt be continued.

[1] *J.A.M.A.*, October 3, 1953.
[2] *Am. J. Dis. Child.*, May, 1955.

THE FERTILIZING CAPACITY OF SPERMATOZOA.

AN investigation of sterility in the male usually consists of an examination of a sample of semen to determine the concentration of spermatozoa and the percentage of those that are normally formed and have normal motility. However, some recent work indicates that even normal spermatozoa have to undergo a specific process of development before they can fertilize an ovum. M. C. Chang[2] has reported on the development of the fertilizing capacity of rabbit spermatozoa. His technique was to induce ovulation by the intravenous injection of sheep pituitary extracts, and then to deposit spermatozoa in the Fallopian tubes; later the tubes were washed out and the ova examined. Chang found that spermatozoa obtained from the epididymis, or washed from the vagina immediately after ejaculation, or washed from the uterus within four hours of ejaculation, were incapable of fertilizing ova, but spermatozoa which had been in the uterus or tubes for some twelve hours were fertile. Other workers had shown that while freshly ejaculated sperms could not fertilize ova *in vitro*, sperms that had been washed from Fallopian tubes twelve hours after mating could do so. It seems, therefore, that spermatozoa require a certain length of time either in the uterus or in the tubes to develop their fertilizing capacity, and there is evidence that spermatozoa which have not gone through this development have not the power to penetrate the *zona pellucida*. Whether a similar phenomenon takes place in man is not known, but it is possible that some of the hitherto inexplicable cases of human sterility might be explained by some interference with a similar developmental process.

[1] *J.A.M.A.*, June 18, 1955.
[2] *Nature*, June 11, 1955.

Abstracts from Medical Literature.

PÆDIATRICS.

Bryant's Traction as a Cause of Circulatory Complications.

J. NICHOLSON, R. FOSTER AND R. HEATH (*J.A.M.A.*, January 29, 1955) state that the peripheral blood pressure is significantly reduced in children's legs placed in Bryant's traction. The great factor in interfering with the circulation of the vertical leg is hyperextension of the knee, especially in patients over the age of four years, for whom this method of treating a fracture is definitely contra-indicated. The authors discuss six children with circulatory complications after Bryant's traction for simple fractured femur; five of the six had the circulatory impairment in the uninjured leg. There were three degrees of impairment: (i) ischæmic fibrosis of the muscles of the leg with a residual equinovarus deformity, occasionally mistaken for a lateral popliteal nerve palsy; (ii) as in (i) but with a circumferential band of necrosis of skin and underlying muscles in the calf, which healed with a cuff-like defect from adherent scar encircling the leg; (iii) as in (i) and (ii) *plus* gangrene of the foot.

Household Poisoning.

M. I. RODMAN (*J. Pediat.*, February, 1955) has surveyed the contents of common household chemicals with a view to assessing their danger as poisons. He approached manufacturers throughout America. Some cooperated willingly in giving details of their products. Some could not be contacted. The author has grouped the chemicals as (i) polishes and waxes for furniture and floors, (ii) paint solvents and related products, (iii) cleaning, polishing and bleaching agents for fabrics, metal and glassware, (iv) cosmetic preparations, and (v) a miscellaneous group. Common ingredients are listed and their dangers discussed. The author states that in general the cosmetic group is the only relatively safe one. He advocates the proper labelling of any potentially dangerous preparation.

Early Operation for Myelocele and Meningo-Myelocele.

W. R. CHAMBERS (*Am. J. Surg.*, October, 1954) in 1950 published a report on the operations in 10 unselected cases of *spina bifida*. Most of these cases were of such a nature that there was a question whether or not they would have been recommended for surgery by ordinary criteria. The basis of these operations was to salvage what was possible, and to learn whether or not the criteria which have been generally accepted for operation in the past might not be too strict. It was also desired to test whether very early operation might not have advantages over a long waiting period such as is advocated by others. The immediate results were most encouraging, and seven of these children are living today. Two eventually died of hydrocephalus and one of enteritis. The author reviews the present state of the seven survivors, and the results are excellent. Early operation is advocated for all cases in which infection is a real danger because of a thin covering or because of the possibility of the presence of a dural sinus. The author states that the presence of hydrocephalus or the possibility of a developing hydrocephalus is now not considered by many as a contra-indication to operation. A further consideration is the finding by several authors of factors which would promote progressive neurological deficit later if not found and removed at early operation, such as constricting bands, lipomata and hypertrophied *filum terminale*. An optimistic view and an intelligent and aggressive attack upon this problem which recurs in one in every 1000 births solicits attention.

Gastro-Intestinal Distension in Infants.

A. GERBER (*J. Pediat.*, January, 1955) reviews previous studies of the source of intestinal gas, and presents further X-ray studies of the problem in infants. He states that gastro-intestinal gas is produced from food decomposition, swallowed air and diffusion from the blood-stream, as has been established in previous studies on the subject. The X-ray investigations presented here were carried out to evaluate further the role of swallowed air in gaseous distension. They indicate that air is swallowed mainly during deglutition; little, if any, air is ingested during crying or sucking without swallowing, as is popularly believed. The suggestion that infants normally ingest air during each inspiration has not been confirmed, although air swallowing unquestionably occurs during the first few minutes of life. Further investigation of the behaviour of intestinal gases is indicated, particularly the role of the autonomic nervous system in the production of gastro-intestinal distension.

Brock Operation for Pulmonary Stenosis.

N. A. ANTONIUS *et alii* (*J. Pediat.*, January, 1955) review the results obtained in treating 39 patients with pulmonary stenosis by the method of Brock, which consists of a direct enlargement of the stenotic pulmonary valve or infundibulum. Most of the patients had the tetralogy of Fallot, in which pulmonary stenosis is associated with a ventricular septal defect and some dextro-position of the aorta. Pertinent clinical data are presented and discussed. The correct preoperative diagnosis of pulmonary stenosis with or without a defect of the ventricular septum was made in all cases, usually on clinical grounds alone, without resort to special studies. In the presence of the tetralogy of Fallot a precise delineation of the abnormal internal architecture of the heart could not be made. The authors state that the Brock procedure is the only one available for surgical relief of pulmonary stenosis without a ventricular septal defect. In the treatment of the tetralogy of Fallot the surgeon has a choice of either the Brock or the Blalock procedure. The choice of operation is influenced by the following factors: the presence of valvular or infundibular stenosis, whether the infundibular stenosis is due to a fibrous septum or a long, narrow muscular tract, the presence or absence of a pulmonary artery, the size of the pulmonary artery, and the degree of overriding of the aorta. Unfortunately these questions cannot be answered with sufficient accuracy with our present methods of investigation, and therefore to a great extent the choice of operation is empirical. The mortality of the Brock procedure is high in the tetralogy of Fallot in children below the age of six years. Since in this group a severe form of infundibular stenosis is likely to be present, the Blalock procedure should be carried out. In the older age group of the tetralogy of Fallot, there is more likelihood that the stenosis will be valvular or that infundibular stenosis will be less severe. In this group the mortality rate of the Brock procedure is low and the results are excellent. In this group, therefore, the Brock procedure is the one of choice. In the presence of overriding of the aorta of more than 50%, neither procedure is feasible, and surgery is contraindicated at the present time.

Cerebral Arterio-Venous Aneurysm in the Newborn.

B. K. SILVERMAN *et alii* (*Am. J. Dis. Child.*, May, 1955) report two examples of congestive cardiac failure in newborn infants caused by large cerebral arterio-venous aneurysms. They state that congestive cardiac failure with gross cardiomegaly is very rare in the early newborn period. It may result from gross congenital cardiac defects, chiefly aortic atresia, transposition of the great vessels and true *truncus arteriosus*. It may result from primary myocardial disease or from paroxysmal tachycardia. Finally a group of extracardiac conditions may cause it. This group includes patent *ductus arteriosus*, severe hæmolytic anæmia, multiple cerebral telangiectases and arterio-venous aneurysm. The two infants described had large hearts with loud systolic murmurs, tachycardia and congestive cardiac failure. Autopsy revealed in one a massive vascular network that almost replaced the right cerebral hemisphere and in the other a large cirsoid aneurysm forming a fistula between the middle cerebral artery and the sigmoid sinus. In both, the hearts were greatly enlarged and had a patent *foramen ovale*, but were otherwise normal.

Intestinal Obstruction in the Newborn.

H. H. DIXON (*Arch. Dis. Childhood*, February, 1955) discusses various aspects of intestinal obstruction in the newborn. He states that truly bile-stained vomitus is extremely rare in infancy in the absence of intestinal obstruction. Not infrequently yellow vomitus is produced in the neonatal period, but this is found not to contain bile pigments on testing with Fouchet's reagent. The misconception that bile-stained vomitus is not uncommon in normal newborn infants and the apparent well-being of the baby suffering from intestinal obstruction until secondary vascular changes occur in the involved bowel are important factors encouraging delay in treatment. Plain

radiographs of the abdomen, not barium meal studies, are the only important confirmatory investigations. Care is needed to exclude brain injuries, infection, cretinism, cystic fibrosis of the pancreas and hiatus hernia before laparotomy is performed. Discussing duodenal atresia and stenosis, the author mentions the high incidence of mongolism (nearly 30%) and the possible value of feeding through a transanastomotic tube after operation. Basing his opinion partly on animal experiments of his own, he suggests that improved results may be obtained in jejuno-ileal atresia by resecting the hypertrophied and dilated terminal bulb (usually 15 to 25 centimetres) and then performing "end-to-back" anastomosis. He criticizes the concept that jejuno-ileal atresia results from incomplete recanalization of bowel and suggests that fusion of the walls and cicatrization after injury to the bowel, such as by incarceration of the physiological umbilical hernia with nipping of the parts, is a more satisfactory hypothesis. He states that in pre-operative management the use of gastric aspiration is helpful in preventing respiratory complications. Operation is urgent, and resuscitation must only be started before operation and then continued in the theatre. A transverse supraumbilical muscle-cutting incision is preferred. Plasma should be given regularly post-operatively to minimize protein depletion, but in other respects intravenous therapy should be conservative, nor should oral feeding be hurried.

ORTHOPÆDIC SURGERY.

Arthrodesis of the Hip in Wide Abduction.

L. C. ABBOTT AND D. B. LUCAS (*J. Bone & Joint Surg.*, December, 1954) state that in patients who have suffered destruction of the femoral head and neck following disease or previous surgery, the ordinary methods of arthrodesis often fail. They advocate in these cases arthrodesis of the hip in wide abduction, followed about six weeks later by subtrochanteric osteotomy. They state that abduction arthrodesis provides intimate contact between the raw cancellous surfaces of the denuded greater trochanter and acetabulum. Circulation to the apposed surfaces is excellent. A constant compressive force is maintained by the adductor muscles. When the femur is abducted about 100°, the line of pull of the adductor muscles coincides with the direction of the shaft. As the femur is abducted, tension in the adductors is developed by passive stretch. This passive tension is transmitted axially to impact the femur against the acetabulum, thereby providing excellent contact and immobilization. Tightness of the adductors, which is usually found in hip disease of long standing, thus materially aids in immobilizing the fusion site. Operative procedure is given in detail. A bilateral hip spica cast is applied, extending to the toes on the involved side and to the knee on the uninvolved side. Routine skiagrams are made to check the position in the plaster. Six to eight weeks later the cast is removed and the hip is checked clinically and radiologically. If the fusion site is solid, a transverse subtrochanteric osteotomy is carried out. A new bilateral hip spica cast is applied, and the final position is again checked by skiagrams. This cast is maintained until the site of the osteotomy has healed. Weightbearing is resumed gradually, and shortening is compensated with a heel lift. The average amount of shortening was 3·1 centimetres and varied according to the amount of bone destruction in both the acetabulum and the greater trochanter. If there has been no destruction, the amount of shortening will be about 2·5 centimetres. The authors conclude that abduction arthrodesis provides the following conditions favourable to union: a large area of contact between cancellous-bone surfaces with excellent circulation, firm apposition and good immobilization. Important to the maintenance of contact at the site of fusion is the axial impaction provided by the tightness of the adductor muscles.

Lumbo-Sacral Arthrodesis After Laminectomy.

E. W. O. ADKINS (*J. Bone & Joint Surg.*, May, 1955) states that the usual methods of posterior arthrodesis of the lumbo-sacral joint are not satisfactory in cases in which laminectomy has been performed. Estimation of fusion by mobility radiographs is unreliable and cannot distinguish between fibrous ankylosis and bony fusion. Bone grafts inserted from behind between the vertebral bodies almost invariably fail to become incorporated. Intertransverse arthrodesis has given promising results and is probably the best method available at present. Details of the operation of lateral fusion between the transverse processes are given. The author states that immobilization of the lumbo-sacral region has always presented a difficult problem, and it is doubtful whether any of the usual methods are entirely satisfactory. The only completely satisfactory method of immobilization is a combination of plaster jacket and double short hip spica. The author points out that this presents a formidable nursing problem, and he has used it only on rare occasions. After most of the operations for disk lesions in which arthrodesis was considered desirable but not absolutely essential, the author compromised with a combination of plaster jacket and single short hip spica. The plaster is retained for fourteen to sixteen weeks, and then replaced by a plaster corset or Jordan's brace. Movements of the uncontrolled hip probably cause slight rotation movements of the pelvis within the plaster, with the possibility of slight movement at the lumbo-sacral joint. The author has come to the conclusion that in practice this method ensures better immobilization than a plaster bed. He states that it is possible that the difficulty of securing fusion of the lumbo-sacral joint is largely explained by the impossibility of providing adequate immobilization, particularly when long grafts are used. These often appear to fuse at one end but develop a pseudarthrosis at the other end, where any slight movements are magnified by the lever-like action of the long graft. To minimize this effect, when more than one segment has to be fused, and no matter what type of graft has been chosen, a series of short intersegmental grafts should always be used in preference to a single long graft. Firm impaction of the grafts between the two vertebræ to be fused can itself produce considerable fixation; and while the available methods of immobilization, either external or internal, remain unsatisfactory, this is probably the best safeguard against failure.

Osteotomy as an Aid to Arthrodesis of the Hip.

A. G. APLEY and R. A. DENHAM (*J. Bone & Joint Surg.*, May, 1955) state that far too many patients have persistent pain after arthroplasty; but if on the other hand arthrodesis is successfully obtained, pain is abolished and stability is assured. They point out that the disadvantages of arthrodesis—namely, technical difficulties in obtaining solid fusion, knee stiffness and pain, hip deformity and back pain—can be overcome by the use of high femoral osteotomy as an aid in arthrodesis. The hip is arthrodesed in the deformed position, and the deformity is corrected by osteotomy. The latter procedure also relieves strain on the side of the arthrodesis. Knee range can be readily retained by treating the patient in traction for the first six weeks instead of using plaster. Thirty-three arthrodeses were attempted without osteotomy. Only 13 were satisfactory. The authors found that even in the best method of obtaining fusion—namely, denudation of articular surfaces and fixation with a trifin nail—sound fusion occurred in only seven out of ten cases when not associated with osteotomy. In a series of 23 unselected cases in which osteotomy was performed in addition to arthrodesis, fusion occurred in 22.

Transplantation of the Posterior Tibial Tendon.

M. B. WATKINS, J. B. JONES, C. T. RYDER AND T. H. BROWN, JUNIOR (*J. Bone & Joint Surg.*, December, 1954) state that transplantation of the posterior tibial muscle and tendon through the interosseous space is a valuable operative procedure and can be relied upon to restore active dorsiflexion of the foot. The operation is applicable to a wide variety of conditions in which the posterior tibial muscle has good power and there is a need for an additional dorsiflexor of the foot—for example, poliomyelitis, spastic paralysis, *spina bifida*, relapsing club-foot, peroneal paralysis (traumatic), atypical muscular dystrophy, Friedreich's ataxia and Charcot-Marie-Tooth disease. Posterior tibial transplantation cannot be expected to produce positive correction of a fixed deformity any more than can other tendon transfers. Therefore, any structural equinus, varus or other deformity should be dealt with before tendon transplantation, by corrective plasters, calcaneal-tendon strengthening, subtalar arthrodesis or other procedures as the circumstances indicate, so that the foot can be placed in neutral position with at least 90° of dorsiflexion. The operative technique is fully described. Operation has been performed 29 times on 26 patients; in two cases the anterior tibial tendon was also transplanted to a more lateral position.

Clinico-Pathological Conferences.

A CONFERENCE AT SYDNEY HOSPITAL.

A CLINICO-PATHOLOGICAL CONFERENCE was held at Sydney Hospital on March 17, 1955, the medical superintendent, DR. NORMAN H. ROSE, in the chair. The principal speaker was DR. G. C. WILLCOCKS, an honorary consulting physician to the hospital.

Clinical History.

The following case history was presented.

The patient, a male infant, aged four months, was admitted to hospital with a history of vomiting after each feed for the previous three days. Prior to this he had been well, apart from a slight cough for one week. The child remained alert and not irritable until the night before admission, when he was noticed to have a squint. On the morning of admission he vomited dark red coffee-ground material. He was noticeably pale, appeared to be in pain and could not hold his head up. The pain was not related to micturition. The bowels had opened on the morning of admission, and the stool was normal.

The baby was breast fed only. He had weighed seven pounds at birth and had gained weight normally. His birth history and subsequent development had been normal. There were eight young children in the house, none of whom had had recent illnesses.

Examination of the child revealed that he was pale and lying quietly in bed with the head turned to the left and dark brown stains around the lips. The fontanelles were depressed, no neck stiffness was present, and the extremities were hypertonic with increased tendon reflexes. The right plantar response was extensor and the left flexor. There was some trismus present. The right pupil was larger than the left; both reacted briskly to light. The ocular fundi were normal. Lateral nystagmus was present with the fast component to the right. There was a pin-hole urinary meatus. The bladder was not palpable. The pulse rate was 140 per minute, the respiration rate 40 per minute and the temperature 98·2° F. No other abnormalities were found on physical examination.

Lumbar puncture revealed clear fluid under a pressure of 140 millimetres of fluid; the Queckenstedt response was normal. There were 150 red cells, 31 polymorphonuclear cells and eight lymphocytes per cubic millimetre of fluid. No organisms were seen on smear examination, and no growth was later obtained on attempted culture.

A blood count revealed a hæmoglobin value of 10·3 grammes *per centum*. The red cells showed marked polychromasia, and numerous stippled cells and nucleated red cells were present. The leucocytes numbered 32,000 per cubic millimetre, made up of neutrophile cells 77% (fairly marked shift to the left and slight toxic granulation), metamyelocytes 2%, myelocytes 1%, lymphocytes 15% and monocytes 5%. The pathologist commented that there was evidence of pronounced bone marrow regeneration in addition to toxic irritation of the leucopoietic tissue.

Following lumbar puncture a fit lasting ten to fifteen seconds occurred. There were rapid blinking movements of the eyelids and tonic extension of the left arm, and Cheyne-Stokes respiration followed. An hour later 40 millilitres of dark coffee-ground material were aspirated from the stomach. Three hours after the child's admission to hospital the temperature rose to 101·6° F., and twitchings of the facial muscles were noticed. Eight hours after admission the infant became comatose, and fine twitchings involving mainly the left side of the face and the left leg were noted. The eyeballs were moving jerkily in all directions. Half an hour later the child died after generalized convulsions.

Clinical Discussion.

DR. NORMAN H. ROSE: Ladies and gentlemen, we have as our principal speaker today one of our most distinguished alumni, Dr. George Willcocks, who was associated with this hospital for many years. I suspect that he was chosen to discuss this case partly because of his long association here and partly because of a long association with the Children's Hospital. This case, I might add, has been discussed many years ago, and the commentator and the people who subsequently discussed the case failed to reach the correct diagnosis. I wish Dr. Willcocks more success today. For those of you who are attending this meeting today for the first time, I should like to say that these meetings have reached manhood: today is the twenty-first consecutive monthly clinico-pathological conference. They have been held monthly on the third Tuesday of each month without a break apart from the annual recess in December. We have had some good discussions and I think what we are about to hear today will be well up to standard. I should like to introduce Dr. Willcocks.

DR. G. C. WILLCOCKS: Mr. Chairman, ladies and gentlemen, when I was asked to speak on this case I said yes, because so often I have refused to come to meetings, but I have wondered since whether I was the right one to select. I think there are many here who would give a much better dissertation on the case than I could.

I think you have the paper before you to indicate the history. It is the case of an infant of four months who vomited for three days before admission to hospital, and soon after admission vomited dark red, coffee-ground material, which I should have thought was blood; but, of course, it sounds like a pathologist—he does not commit himself! The past history of this child was that it was born a normal baby, weighing seven pounds, and had normal health until, so far as I can make out, three days before admission to hospital. The child had been breast fed throughout and, I understood, had no ailment except a slight cough a week before admission.

I was asked to say something for the benefit of students as to the approach to the diagnosis in children's diseases. Well, it is really quite a time since I severed my connexion with the Children's Hospital, and I do not know that there are very many students here; if there are, they probably know more about the approach to the diagnosis than I do. But there are one or two things I would like to say with regard to the approach to diagnosis. You have been told, of course, that the important thing in childhood is inspection, to examine the child by inspection; preferably, in my opinion, not looking them in the face, because they always cry when I look them in the face. But "keep away and look at them sideways" is my idea of examining an infant. Well, in these cases, of course, we do not have the chance of examining the child, and to the clinician that is a very serious defect. It may be that we would not know any better if we saw the subject. But on the other hand, as we are brought up to do nothing else but look at patients, I think that if we did see the patient, we might sometimes have a better idea as to the diagnosis.

Again, as to the approach to children, I might mention one or two cases. I will not take up a great deal of time, and I have been told this is not a place for levity, at medical meetings. But I might mention one or two cases. There was a doctor friend of mine who used to call me in to see his children, generally at night time. He would ring up and say: "I am not going to tell you what I think, but so-and-so has been vomiting and has head retraction and Kernig's sign. I wish you would come and see him. I won't tell you what I think." One night he rang me and said: "I wish you would come and see John. He is lying in bed, crying, and he won't let us get near him, and he won't bend his knee—his right knee. I won't say what I think, but that's the position. He seems to be in intense pain." Well, of course, I was younger than, and I always thought of the worst. I thought immediately of osteomyelitis in this case. I went over, and the boy was certainly in bed, crying, and would not move his knee. But he did not look very ill. So I offered him threepence to move it. And he moved it at once.

Now that, I think, very often applies with children. When I used to examine crying children in the wards I am afraid my psychological approach was very bad. I always gave them threepence. I should think, with young practitioners, the sum would have gone up with inflation, and you would have to offer them a shilling or perhaps two shillings. Two shillings might be better because they would not swallow it.

Some further history about this child—and I hope someone will correct me if we omit anything important. We have the history of the onset with vomiting, and vomiting blood, and then we have the past history, which is normal, and then we have the examination, which is very adequately set out, except that some things are not mentioned. The child was flaccid and pale with head turned to the left, and there was rigidity of the limbs with increased reflexes, and nystagmus with the fast component to the right, so the slow one would be to the left. The child did not have head retraction, but it was very pale. The right plantar response was extensor, and the left was flexor. The normal response in an infant is extensor, so one would think that the flexor response on the left would be a sign of a lesion on the right side of the brain. The child had nystagmus, was stuporose or partly conscious, and had a dilated right pupil, rigid limbs and increased reflexes, and I think the head was

turned to the left. All that is very puzzling, as to the situation of the lesion. The nystagmus having a slow component to the left should indicate a lesion on the left side, but the down-going toe on the left should indicate a lesion on the right-hand side. So I assumed that there was an extensive cerebral lesion. It seemed to affect the cerebellar fibres on the left-hand side and the motor tracts on the right-hand side. I am not really going to try to locate that lesion now, except to say that it is so extensive that it seems it must be involving the middle part of the brain, perhaps the brain stem, as well as the cerebellum. When the child was lumbar punctured there were convulsions and twitchings of the left leg and face. Now that might not be very important in a child, but it might indicate a lesion in the cortical or subcortical area on the right side.

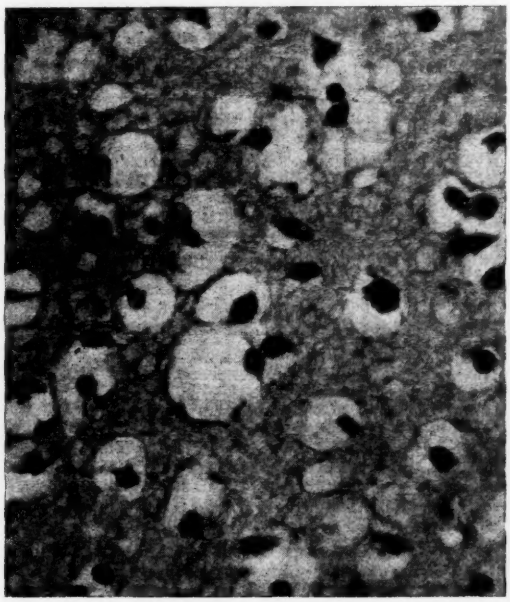

FIGURE I.

Section of brain showing changes of acute lead encephalopathy: cerebral œdema, prominent pericellular clear zones and oligodendroglial swelling.

The lumbar puncture yielded 150 red cells, 31 neutrophile cells and one or two lymphocytes. That is a curious discovery. When I first read this history, I thought the child probably had a cerebral hæmorrhage, perhaps from an aneurysm, but when you find only 150 red cells and 31 neutrophile cells per cubic millimetre, it puts you off that diagnosis. There was no great increase in pressure of the cerebro-spinal fluid, and the result of the Queckenstedt test was normal; no growth, a clear fluid, no sign of any infection in it.

Next comes the blood count, which shows low hæmoglobin content, about 10 grammes, and 32,000 leucocytes with 15% lymphocytes, 2% metamyelocytes and 1% myelocytes. We have a history of a marked cerebral disturbance, vomiting of blood and this anæmia, which does not seem to me to fit in with anything very well. It surely is not the anæmia of blood loss, from the vomiting of blood. I thought at first it might be leuchæmia with leuchæmic deposits in the brain. It is certainly not a typical picture of leuchæmia, but it could come within the range of this disease. Then I thought of generalized *lupus erythematosus*, which gives rise to very strange and widespread disturbances. This is a condition which we never or seldom diagnose *ante mortem*.

Well, to consider the cerebral lesion, I thought of the possibility of abscess, giving rise to the neutrophile cells in the spinal fluid, a rapidly developing abscess. I cannot think of any tumour which would give rise to the blood condition, but I would like to ask what was found in the examination of the throat and mouth. Can we know that?

DR. P. FRANCIS: They were normal.

DR. WILLCOCKS: It mentions some reddish stains on the mouth of the child, and I thought with vomiting blood and these stains there might be something wrong with the gums, and that leuchæmia might explain the whole condition. But we have not any real evidence that the condition was leuchæmic. Well, we can approach the diagnosis, I think, by going through the systems in order.

This leucocytosis with a shift to the left makes one very suspicious of an infection. We have not any indication whatever where the infection might be. A slight cough for a week: well, if the child had bronchiectasis, or a lung abscess, that might have explained this condition in the brain. But a slight cough for a week would scarcely give rise to such a marked secondary condition in the brain in a few days. I went through the various systems in order, as they come in the text-book. Infectious diseases: considering the ordinary infectious diseases of childhood, we have no evidence of any of these being present in this child—no rash, no contact with any other child who was ill. The only infectious disease I could think of was a septicæmia, and we have no evidence of its existence or of where it might have arisen. So, I cannot decide on any infectious disease. I thought of infective endocarditis secondary to a congenital heart lesion. But it is a very short history.

Taking the gastro-intestinal tract, I thought of a gastric ulcer, which, of course, is rare in childhood, as a cause of this vomiting, but it would not cause the cerebral sypmtoms. And I thought of a growth somewhere in the abdomen, in the stomach or elsewhere, giving rise to secondary deposits in the brain. But that would not account for the neutrophile cells in the spinal fluid. Considering the respiratory

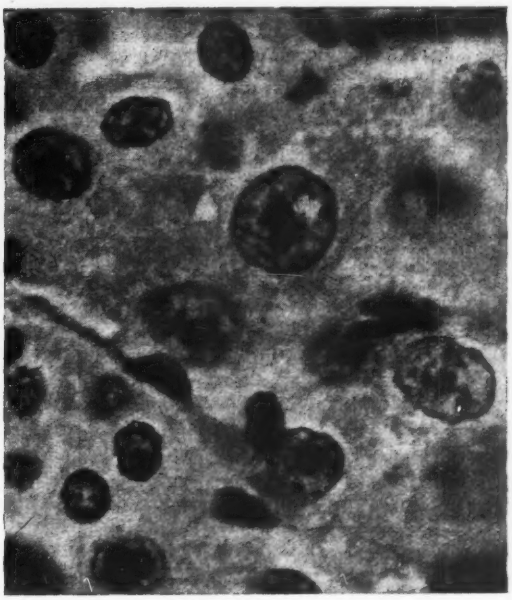

FIGURE II.

Acute lead poisoning. Renal cortex showing nuclear inclusions in tubular epithelial cells. Some of the affected nuclei are enlarged.

system, we have no evidence at all. A growth in the lung could give rise to very similar symptoms, but I find it difficult to account for the vomiting of what I take to be blood. Similarly, lung abscess or empyema or bronchiectasis could have caused a condition similar to this, but we have no evidence of it.

Conditions of the genito-urinary tract: we are told the child had a pin-hole meatus, and I immediately thought of pyelonephritis, which is a common cause of undiagnosable complaints with widespread symptoms. The urine was not examined, but then the bladder was not full, or enlarged, apparently. I was thinking of pyelonephritis superimposed on a congenital malformation, but I thought we had insufficient proof of such a diagnosis. And I do not see how

pyelonephritis would account for the vomiting of blood, if it was blood.

Other things I thought of were poisons. Many poisons could give rise to such a condition, I think, but we have no evidence that any poisons were discovered. They are too numerous to mention, the poisons which a child could be affected by in this way. We have no history of any treatment with drugs which could give rise to the condition, and we have no grounds for believing the child was poisoned by its parents, for instance.

The bones must be considered. A growth in the bones could give rise to changes, secondary deposits in the brain. An embryoma of the kidney, as it used to be called, might give rise to secondary deposits in the brain. Now to come more to fantasies, there is always avitaminosis, but a breast-fed child at four months I scarcely think could have avitaminosis. Then there is purpura, but this is not purpura in the ordinary sense. To consider conditions of the nervous system, of the brain, there is hæmorrhage from a congenital aneurysm, but this would not account for the condition of the blood. I could not think of any cerebral tumour which would cause such a blood condition. Abscess—I could find no reason for an abscess, and there certainly was no meningitis, or, I should think, encephalitis. I thought of a congenital condition like porencephaly, but that would not account for the blood condition or the vomiting of blood. Hodgkin's disease gives rise to a widespread condition, but this blood count and the condition of the brain do not suggest this disease to me, and there were no glands. I thought of ear infection giving rise to secondary abscess in the brain.

In the cardio-vascular system I thought of infective endocarditis. I have mentioned the collagen diseases—disseminated *lupus erythematosus, polyarteritis nodosa*—which always seem to be mysteries and hard to diagnose. Of the blood dyscrasias there is leuchæmia; I do not think aplastic anæmia could cause this cerebral disturbance, nor could a hypoplastic anæmia. So I am really brought to thinking that it was probably an infection and, in the absence of any evidence, that the most likely situation would be in the heart.

I would like to ask somebody else to discuss this.

DR. ROSE: Would you care to ask the opinion of any of the specialists of the results of these special tests?

DR. WILLCOCKS: Well, I would like to know what the opinion of the hæmatologist is on the blood condition.

DR. ROSE: Would Dr. Walsh care to comment?

DR. R. J. WALSH: I have been very interested in this case, but find the hæmatological findings anything but easy to interpret. First I should like to emphasize that at the age of four months the hæmoglobin value of a child is at its lowest level. A child is born with a high hæmoglobin value, but it rapidly declines to reach its lowest level about four months later. It remains fairly constant till its first year of life has been completed, and then it gradually increases till puberty. Actually, in a large series of children we found that the mean hæmoglobin value was about 12·5 grammes *per centum* at four months of age. Now I do not really think that a value of 10·5 grammes *per centum* is indicative of anæmia. Of all children who are allegedly normal and breast fed, 10% to 15% are at that figure or less.

I feel that I am not competent to suggest a diagnosis on the basis of this blood count, but the thought which occurred to me about the child generally was that it could conceivably be suffering from lead poisoning. I know there is nothing in the history to suggest how this child could have obtained lead, unless, perhaps, one of its older brothers (I notice there were eight young children in the house) was interested in fishing and supplied a sinker to the child. A leucocytosis of 32,000 cells per cubic millimetre is somewhat high for such a condition, but acute lead poisoning is nearly always accompanied by a leucocytosis in which the neutrophile cells predominate. I think that such a localized irritation could result in mucosal hæmorrhage in the stomach to account for the vomiting. The stippling of cells which was noticed here is a fairly rapid accompaniment of acute lead poisoning. The fact is also mentioned that there is a marked toxic irritation of the white cells, which could also be explained on the basis of lead poisoning.

DR. ROSE: Would you care to call on any other specialists, Dr. Willcocks?

DR. WILLCOCKS: I should like to hear some comments on the cerebro-spinal fluid.

DR. ROSE: Dr. Tebbutt, would you care to answer that question?

DR. A. H. TEBBUTT: I was very pleased to find Dr. Willcocks in the throes of the neurological story, because I was still more puzzled by that than he was. As regards the blood picture, I am in complete agreement with Dr. Walsh that it does show effects of some sort of poisoning, of which lead is the commonest. It is possible that the child got hold of some sort of lead preparation. I do not see what else could explain the blood picture. The leucocytosis is rather high, but perhaps this is so because it is in a child. I have had no experience with children, but leucocytosis in lead poisoning has been reported. As for the subarachnoid space, and the presence of a considerable number of red cells and white cells there, I could only think of the white cells there being an expression of the leucocytosis in the blood-stream and that any disturbances of the central nervous system might further increase the number of neutrophile cells in the subarachnoid space. The diagnosis of lead poisoning seems to me to be the most logical.

DR. ROSE: We have representatives here today of Sydney Hospital students, residents and honorary physicians. I can see a fair sprinking of people interested in this case who have come all the way from the Children's Hospital and also several representatives from the University, but we will hear from them later. Are there any of the resident medical staff who have any suggestions? Dr. Michell?

DR. G. MICHELL: I am acquainted with the case.

DR. ROSE: Dr. Hudson, would you care to offer your opinion?

DR. C. B. HUDSON: Having attended most of the clinico-pathological meetings which have been held in Sydney Hospital, I think I was present when this case was discussed some years ago.

DR. ROSE: Dr. Robertson, have you an alibi?

DR. T. I. ROBERTSON: Yes, I have an alibi also. I was on duty the night this case was admitted to hospital.

DR. ROSE: I see in the back of the room Dr. Robertson, who was a distinguished member of our staff for some years and who has now allied himself to the Children's Hospital. Dr. Robertson, I see you have been making copious notes.

DR. S. E. J. ROBERTSON: Mr. Chairman, having seen this sort of case, and tried to diagnose it during life, I can well sympathize with Dr. Willcocks. To diagnose the cause of an illness in such a small infant one needs sometimes a crystal ball. During Dr. Willcocks's discussion I made notes approaching the case in a somewhat different way. I thought the diagnosis could best be obtained if one studied the causes of the main clinical feature, convulsions, with secondary factors of vomiting and a rather unusual blood picture. If one goes through the causes of convulsions in a child of this age, the diagnosis may be approached more closely than by using any other method. I think there are no reasons to suspect *status epilepticus* due to idiopathic epilepsy, for there is no family history of epilepsy and no previous attacks. Hypocalcæmia, like rickets, is extremely unlikely in a breast-fed baby. Rare causes of hypocalcæmia, such as hypoparathyroidism, are unlikely.

The convulsions could be due to meningitis or encephalitis, but we have no reason to suspect either. Dr. Helen Walsh, on my left, far too reticent to get up, has suggested tuberculous meningitis to me, which is fairly probable. However, the blood count is not consistent with a brain infection and the cerebro-spinal fluid is not suggestive. The red cells, I think, are due to local trauma. No protein level has been given. There is no report of a Mantoux reaction, no family history of tuberculosis and no chest X-ray film. Thrombosis of cerebral veins is a possibility but undiagnosable with the known facts. Uræmia, with hypertensive encephalopathy, as mentioned by Dr. Willcocks, occasionally causes *status epilepticus*, but there is nothing in the story to suggest it.

Poisons likely to cause such a story are aspirin, camphor, strychnine and iron. The antihistamines cause necrosis of the stomach and convulsions, but were probably not in use when this child had its illness. Without a history of ingestion it is difficult to make a diagnosis of poisoning.

Acute porphyria is extremely rare, but it does turn up occasionally. Now that the Watson-Schwartz test is available, a simple test for urinary porphyrins, this diagnosis may be made more frequently. One was a case of *status epilepticus*, due to acute porphyria, which was diagnosed at the Children's Hospital recently, shortly before death. Acute necrosis of the liver with hypoglycæmia may cause *status epilepticus* before jaundice is detected. Idiopathic hypoglycæmia of infancy may cause severe convulsions and is a reason for estimating the blood sugar in all obscure cases.

I agree with Dr. Walsh and Dr. Tebbutt in that the most likely diagnosis is lead poisoning. The blood picture is hardly likely to have any other cause in a child. Lead poisoning often presents as vomiting followed by severe convulsions. The most difficult point, however, is how could a child of four months of age swallow lead? A registrar at the Children's Hospital confronted with a child with severe convulsions in whom there is a normal cerebro-spinal fluid, apart from an increase in protein, always thinks of lead. These cases, however, are nearly always in an older age group, the age group from eleven months to three years, where dirt eating is quite common and lead compounds are likely to be swallowed. This baby was four months, an age when he could not put anything in his mouth and could only just lift his head. I can see no possible manner in which lead could have been absorbed.

DR. ROSE: Are there any more comments before we ask Dr. Palmer to tell us the diagnosis? Dr. Berns?

DR. W. BERNS: I was interested in the central nervous signs of this infant—the squint, the nystagmus to the left, the altered plantar response. The lumbar puncture findings are difficult to interpret, but the alterations of the polymorphs and lymphocytes are abnormal, although on culture no growth of organisms was obtained. I think the depressed fontanelle must be due to dehydration, as the infant had been vomiting for several days. Some of the manifestations, such as the rapid pulse, the pale appearance and vomiting, I think would be due to the effect of a severe illness. The dark-stained vomitus may be due to superficial mucosal ulceration, perhaps also a manifestation of severe infection. I think perhaps the blood count, which shows a neutrophile leucocytosis, would point to a pyogenic infection. I do not know whether this cough of one week's duration would have any significance unless it was due to a pneumonia, which presents a difficulty in diagnosis in an infant without an X-ray examination of the chest. But I think a possible diagnosis would be meningitis. In view of the fact that the child died very rapidly, I think another possibility is the Waterhouse-Friderichsen syndrome in which the adrenal glands are involved, in a fulminating meningitis. It might explain the rapid demise.

DR. P. ANDERSON: In relation to lead poisoning, which has been canvassed. I cannot imagine it would happen in this enlightened country at this stage of this century, but early in the century there were records of young babies suffering from acute lead poisoning due to the absorption of lead through the skin from dusting powders contaminated with white lead. That is one possible source. My other comment is that cerebral tumours of considerable size have been seen in children without giving rise even to the classical triad of symptoms of increased intracranial pressure until hæmorrhage into them has occurred. I think if we had any of the pathologists here from the Children's Hospital they could tell us of more than one such instance. Squint was one of the first signs noticed in this story, and I feel that possibly there could be an intracranial lesion of a space-occupying nature.

Pathology Report.

The following report was given by Dr. A. A. Palmer, and slides were shown.

The body was that of a well-nourished child of four months of age. The only organs showing macroscopic abnormalities were the liver and stomach.

Liver.—There was evident fatty change; the peritoneal and cut surfaces being pale yellowish pink.

Stomach.—In the stomach contents there was a cylindrical rolled piece of lead foil, about half an inch long and rather less in diameter. The mucosa near the pylorus showed some brown discoloration. A little altered blood was present in the small intestine.

Microscopic Examination.

Brain and Spinal Cord.—There is conspicuous œdema, which is in places pericellular. The appearance closely resembles that illustrated in cases of lead encephalopathy.

Kidney.—Intranuclear eosinophilic inclusions such as occur in poisoning with lead and some other metals are present in many of the epithelial cells of the convoluted tubules. The affected nuclei are often enlarged and irregular in shape.

Liver.—There are intranuclear inclusions in some of the parenchymal cells, but they are smaller and less numerous than in the kidney. There is considerable fatty change.

Spleen, Lung, Thymus.—No significant change.

Final Diagnosis.

Acute lead poisoning with encephalopathy.

Discussion.

DR. ROSE: You will agree that today you have seen a very fine product of the photography department of Sydney Hospital. We have several pathologists here today, and as we have more time left than usually is the case, perhaps we could have some discussion about these pictures.

DR. TEBBUTT: I thought that the red cells were probably getting out into the perivascular spaces and lymphatics in the same way as the leucocytes.

PROFESSOR F. R. MAGAREY: I was rather hoping that Dr. Anderson was going to mention the old-fashioned lead nipple shield, and I suppose the history was gone into as an afterthought to find whether the child had in fact ingested portion of such a shield. I should like to criticize part of Dr. Palmer's description of the second slide purporting to show pericellular œdema, because I do not think it did show such œdema. What it did demonstrate, however, was acute oligodendroglial swelling. The little black nuclei surrounded by the stainless vacuoles are oligodendroglia, and we know that oligodendroglia, when photographed by the movie camera, can be seen to blow up to the size we saw in the section and then squirt something out somewhere and go back to their normal size. This histological change is not specific for acute lead poisoning, because we see it in almost any case in which there have been fits or convulsions of some kind. I am not questioning the diagnosis at all, for there were bubbles of interstitial œdema as well, which were not related at all to the oligodendroglia, and the acute oligodendroglial swelling was a good deal more marked than we usually see in other cases of convulsions.

DR. ROSE: I am sure if Dr. Willcocks had used his well-known Sherlock Holmes power of observation he would have noticed in the audience today Dr. Gordon Smith, who is now Sydney's foremost toxicologist. He was once a member of the pathology department of Sydney hospital and is now at the School of Public Health. Dr. Smith was informed of the diagnosis in this case and was invited to come along to round off this discussion.

DR. G. C. SMITH: I must admit that in this case I feel on rather unsteady ground, because most of the cases of lead poisoning with which I have to deal are industrial in origin, and more or less chronic in type. I do not think I have seen lead poisoning in a child in recent years, and acute lead poisoning in industry is very rare. I remember one case, and that was a man who had been exposed to vapour of lead tetraethyl. He had been sent in to clean out a storage tank and was exposed to a heavy concentration of vapour from this organic lead compound.

As Dr. Rose said, I had been informed of something of the diagnosis in this case beforehand, and to me it has been very interesting to hear all that has been said. I suppose lead encephalopathy is about the only condition which could account for both the cerebral and other features. But there are one or two points which do not fit in with the usual picture of lead encephalopathy. I think most investigators report that the cerebro-spinal fluid pressure is raised and may be even of the order of 600 or 700 millimetres of water. Also, the increase in the cell count is usually only slight and mostly involves the lymphocytes. Cell counts higher than about 60 per cubic millimetre do not appear to be common. So far as the cerebral lesions themselves are concerned, some authorities believe that there is intense cerebral œdema and that the signs and symptoms are primarily due to increased intracranial tension. The hæmatological findings also are quite interesting. They are not typical of those we usually obtain in lead poisoning of industrial workers, which is due to inhalation rather than the swallowing of lead compound. In this case the pathological condition probably commenced before the nipple shield was swallowed—that is, presuming the lead foil was from a nipple shield. This is hard to prove; but if we assume it was, probably the child would have absorbed small quantities of lead for some period prior to the actual swallowing of the lead foil. It would be very interesting to know just what the interval was between the swallowing of the lead foil and the onset of symptoms. I suppose we have no means of ascertaining that; but whatever the period was, the total dosage of lead in relation to the size of the infant must have been quite appreciable to produce the symptoms and signs which have been revealed. With regard to treatment, reports indicate that the administration of disodium calcium ethylene diamine tetraacetate would be justified in cases such as this.

DR. ROSE: I should like to emphasize one lesson which arises from today's case—that is, that this problem is not an uncommon one. Two cases have occurred in Sydney Hospital since the admission of the one we have just discussed in which the patient has suffered from a piece of lead in the stomach. The diagnosis in both cases has been made by the resident medical staff, who were made aware of the condition by a previous discussion of this case. I think if we have made more popular a knowledge of the existence of such a condition we have achieved something worth while.

Diagnosis.

Acute lead poisoning with encephalopathy.

British Medical Association News.

NEW SOUTH WALES FEDERATION OF COUNTRY LOCAL ASSOCIATIONS.

FOURTH ANNUAL POST-GRADUATE COURSE.

THE fourth annual post-graduate course of the New South Wales Federation of Country Local Associations was held at Wollongong from March 14 to 18, 1955, under the presidency of DR. B. A. COOK (Bulli).

The Medical History of the Wollongong District.

DR. H. H. LEE (Wollongong) read a paper entitled "A Medical History of the Wollongong District" (see page 353).

The Roy D. Mulvey Memorial Oration.

DR. B. A. COOK (Bulli) delivered the Roy D. Mulvey Memorial Oration (see page 356).

Recent Advances in Arthritis.

DR. L. J. A. PARR (Sydney) read a paper entitled "Recent Advances in Arthritis" (see page 358).

DR. R. B. PILCHER (Gunnedah) asked whether there was a relationship between rheumatoid arthritis and psoriasis, and whether the two diseases occurred coincidently in the same patient.

Dr. Parr, in reply, said that there was a relationship between rheumatoid arthritis and psoriasis, as the diseases waxed and waned together. The terminal joints, which were rarely involved in rheumatoid arthritis, were commonly involved in rheumatoid arthritis associated with psoriasis. Some pathologists had suggested that the pathological condition in the joints was different from that of the classical disease. Dr. Parr said that in his experience the type of psoriasis associated with rheumatoid arthritis was of two varieties—one tended to disappear under the action of the sun, and the other was unfavourably affected by sunlight. In the former case he gave large doses of vitamin D_2, while in the latter arsenic was valuable. In both types the joint lesions paralleled the amelioration of the psoriasis. Dr. Parr said that he had also found gold of value in such cases.

The Use of Antithyroid Drugs.

SIR HUGH POATE (Sydney) read a paper entitled "The Use of Antithyroid Drugs" (see page 363).

Dr. R. B. Pilcher asked whether the estimation of the serum cholesterol content was of use in the diagnosis of thyrotoxicosis.

Sir Hugh Poate, in reply, said that the estimation of the serum cholesterol content was not in itself very helpful. There was a wide variation in the normal figures. Although the level tended to fall in thyrotoxicosis, the test could be regarded only as an ancillary aid, to be used in conjunction with the estimation of the basal metabolic rate and the clinical condition of the patient.

DR. F. W. WALTON (Wollongong) asked two questions. The first was whether a therapeutic trial of iodine was as good as a similar trial of thiouracil in the diagnosis of thyrotoxicosis. The second was whether small maintenance doses of thiouracil given from the beginning of treatment, as advocated by Isbister, were satisfactory.

Sir Hugh Poate, in reply to Dr. Walton's first question, said that he did not think that the therapeutic trial of iodine was as satisfactory in diagnosis as the similar use of one of the thiouracils. In order that the trial should be carried out efficiently, the patient would have to spend three weeks in bed in hospital under peaceful conditions. During the first ten days the patient would be given regular sedation with glucose and probably vitamins of the *B* group. The pulse rate would be recorded every four hours. For the next ten or twelve days the patient would be given three drops of Lugol's iodine three times a day. If at the end of the three weeks there was a decided drop in the pulse rate, the patient had gained in weight and the symptoms had lessened, then the diagnosis of thyrotoxicosis could be made with certainty. When thiouracil was used instead of iodine, there was no need for the patient to remain in bed, since with the correct dosage the patient with thyrotoxicosis would show a definite response, which would establish the diagnosis. The dosage of thiouracil was 0·1 gramme three times a day for one week, then 0·2 gramme three times a day for two weeks; after that the dose was reduced to 0·1 gramme three times a day. In reply to Dr. Walton's second question, Sir Hugh Poate said that in his experience doses of thiouracil smaller than those he had mentioned tended to mask the condition, the response was very slow, and there seemed a greater tendency to the development of frustration hyperplasia in the gland. The essential aim of treatment was to secure rapid control of the level which would maintain the patient's improvement.

DR. F. M. CLOUSTON (Roseville) asked whether iodized salt was of any value in the prophylaxis of the diffuse "goitre" of puberty and of the adolescent girl.

Sir Hugh Poate, in reply, said that iodized salt was of use. Once the thyroid had enlarged, the patient could be given dry thyroid. The avoidance of vegetables of the cabbage family was advisable.

DR. C. A. HOLMES (Inverell) asked what was the treatment of a person suffering from a non-toxic nodular goitre who was otherwise well.

Sir Hugh Poate, in reply, said that if a multinodular goitre developed on a simple colloid condition of many years' standing, and if the condition became evident between the ages of thirty-five and forty-five years, the patient should be advised to submit to operation, as a toxic condition was apt to supervene about the time of the menopause. However, if a single tumour was present, it should be removed at any age, and particularly if the patient was a male, as such tumours were prone to become malignant.

DR. B. A. COOK (Wollongong) asked Sir Hugh Poate to comment on a statement by a leading American endocrinologist that at one clinic there had not been a thyroid operation for a period of five years.

Sir Hugh Poate, in reply, said that he thought that the clinic to which that statement applied was one where thyrotoxicosis was treated with radioactive iodine isotope. Apparently the workers there did not take into consideration the fact that a large gland might still persist. Moreover, that method of treatment had not been in use sufficiently long for it to be determined whether carcinoma was more likely to supervene on a diseased gland treated in that way.

Pulmonary Conditions in Miners.

At the Wollongong District Hospital papers were read by Dr. K. G. Outhred, Dr. R. G. Epps and Dr. M. J. Flynn, and the following three demonstrations were given: (i) methods of pulmonary function testing in use at the Pulmonary Clinic at the Wollongong District Hospital; (ii) physiotherapeutic methods, including the treatment of bronchiectasis; (iii) several types of oxygen and aerosol therapy.

The Treatment of "Dusted" Miners.

DR. K. G. OUTHRED (Joint Coal Board, Wollongong) read a paper entitled "The Treatment of 'Dusted' Miners at the Wollongong Pulmonary Clinic" (see page 365).

Aspects of Diagnosis in Chronic Bronchitis and Emphysema.[1]

DR. R. G. EPPS (Sydney) read a paper entitled "Aspects of Diagnosis in Chronic Bronchitis and Emphysema" (see page 367).

Pulmonary Function Testing.

DR. M. J. FLYNN (Sydney) read a paper entitled "Wollongong Pulmonary Clinic: Pulmonary Function Testing" (see page 368).

[1] The diagnostic criteria adopted at the Wollongong Pulmonary Clinic were set out at some length; they are not reproduced here owing to lack of space.

DR. B. A. COOK, in opening the discussion, stressed the rather high prevalence of chronic respiratory diseases in the Greater Wollongong area. He referred to his trip overseas some years previously, during which he had visited the Pneumoconiois Research Centre in Cardiff and various similar centres in the United Kingdom, and said that in his opinion the work of the Pulmonary Clinic at the Wollongong District Hospital was well up to their standard. Dr. Cook then welcomed to the meeting Dr. W. E. George, Chief Medical Officer of the Joint Coal Board, and suggested that he might assist in the answering of questions.

DR. E. L. MILGROVE (Medical Officer, Australian Iron and Steel Proprietary, Limited, Port Kembla) asked why the incidence of pneumonokoniosis had always been greater in the southern than in the western and northern coalfields of New South Wales.

Dr. George, in reply, said that in his opinion one of the most important of the various factors responsible was the very dry nature of the coal seam commonly worked in the southern district, as compared with other areas. The question was largely one of the quantity of dust which remained in the atmosphere inhaled by the men underground.

A second question asked was why it often happened that the postural treatment of bronchiectasis carried out by the general practitioner did not lead to such good results as those achieved in hospital clinics.

This question was answered by Miss McKay, a senior physiotherapist in chest diseases, who had attended to assist with the demonstration. Miss McKay said that two things were important in postural drainage. Firstly, the correct positions for posturing varied with the areas of lung involved, and the posturing had to be carried out in relation to the extent and location of disease present in the particular patient. Secondly, it was imperative for the patient to cough and expectorate as much sputum as possible during each posturing, and he should be encouraged to do so. Miss McKay believed that if careful attention was given to those points, uniformly good results would be obtained.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on March 16, 1955, at the Queen Victoria Memorial Hospital, Melbourne. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital.

Hæmorrhage during Labour due to Blood Incoagulability.

DR. WINIFRED KENNAN presented two patients who had suffered from hæmorrhage during labour due to incoagulability of the blood. The first patient was a *primigravida*, aged twenty-seven years, who had been in hospital when fourteen weeks pregnant with an attack of infective hepatitis, and again from the thirtieth week with signs of toxæmia of pregnancy (excess weight gain, œdema of the feet and a trace of albumin in the urine). Labour was induced at term by administration of castor oil, enema, stripping of the membranes and intravenous "Pitocin" infusion. Fifteen minutes before delivery of a stillborn female infant the fœtal heart rate dropped to 100 per minute, and then to 80. Copious bleeding followed delivery and was thought, at first, to be due to a vaginal tear, which was sutured, but it continued steadily after expression of the placenta eighteen minutes after the delivery. It was noticed that the blood did not clot, and that there was a continuous ooze from the vaginal well. Blood transfusion was commenced, and the blood did not clot until five pints had been given. There was a period of shock before transfusion was started, and slow improvement took place over the next two hours. For one week after delivery there was extreme oliguria, and further transfusions were given on the third, fifth and twenty-first days. Her bleeding tendency was considered to be due to lack of fibrinogen, and the stillbirth to a concealed antepartum hæmorrhage.

The second patient was a *multigravida* who had had four children, showing a condition of afibrinogenæmia occurring in an Rh-negative patient, with intrauterine death and retention of the fœtus *in utero* for some weeks. The patient had suffered from *hyperemesis gravidarum* in early pregnancy and again at twenty-seven weeks, when she showed signs of liver damage (reduction of total serum protein content, reversal of albumin/globulin ratio and positive results from cephalin flocculation test). Labour commenced spontaneously at the thirty-sixth week. (Fœtal death had been diagnosed some weeks previously.) There was a history of spontaneous bruising for two weeks and steady hæmorrhage before admission to hospital. That continued, and it was noticed that the blood did not clot. After delivery of a macerated fœtus soon after the patient's admission to hospital, the patient continued to bleed in spite of treatment (intravenous administration of ergometrine 0·5 milligramme, intramuscular injection of calcium gluconate 10 millilitres, and injection of vitamin *K* 60 milligrammes).

Blood transfusion was commenced immediately, but no clotting occurred, and two hours after delivery administration of fibrinogen was begun (five grammes—each gramme dissolved in 50 millilitres of distilled water). After receiving the first gramme, the patient was shocked, so a further pint of blood was "pumped" in, with a subsequent increase in blood pressure. The remainder of the fibrinogen was then given. After administration of two and a half grammes, the blood clotted in six minutes, but the clot redissolved in half an hour.

After administration of five grammes (two hours from the start) the clot did not redissolve. The total blood lost was five pints, and the blood replaced was three pints. The patient was discharged from hospital one week later, but returned in a week with hæmatemesis. The clotting time was normal, but the hæmoglobin value was 65%, and, after a transfusion, the patient quickly improved; she was discharged from hospital two weeks later.

The patient's previous baby, born in 1952, had required a replacement transfusion, and blocking antibodies (1:250 titre) were present throughout the pregnancy.

Dr. Kennan discussed the condition of afibrinogenæmia and said that it might manifest itself in three conditions complicating pregnancy: (i) *abruptio placentæ*, in which rupture of the basal decidual plate occurred, resulting in the liberation of thromboplastins into the maternal circulation; (ii) amniotic fluid embolism—amniotic fluid was a rich source of thromboplastins, which were derived normally from fœtal exfoliation, and also from the decidua; (iii) rhesus incompatibility resulting in intrauterine death and retention of the fœtus *in utero*—the fœtal membranes and decidua disintegrated and allowed the absorption of thromboplastins into the maternal circulation.

Dr. Keenan said that the present hypothesis was that, in such conditions, the thromboplastins caused extensive intravascular clotting, resulting in a marked depletion of the circulating fibrinogen, which the liver was unable to make good in time to prevent the hæmorrhagic diathesis from manifesting itself. It was suggested that a fibrinolysin was produced as a counter measure and that that tended to aggravate the condition by producing fibrin emboli.

Diagnosis might be made by the "clot observation" test. The possibility of the condition must be borne in mind in the three classes of patients quoted, and administration of pure human fibrinogen was essential, should severe afibrinogenæmia develop, if life was to be saved. Restoration of blood volume was also of prime importance and must be effected, in addition, to combat shock.

Dr. Kennan showed also a uterus with *placenta accreta in situ*, and microscopic slides of the specimen. The patient was a *multipara*, aged thirty-eight years, who had had six children. She had had manual removal of the placenta and blood transfusions with each delivery, and the placenta had been found to be extremely adherent at the last delivery in 1949. She was treated by Cæsarean hysterectomy, and a living male child delivered. The placenta appeared spread out over most of the inner surface of the uterus, and on incision across the fundus no line of cleavage between the placenta and uterus could be seen. Microscopic section showed no identifiable remains of endometrial glands, the placenta being directly continuous with the uterine wall.

Dr. Kennan said that the essential cause of *placenta accreta* was a partial or complete absence of the decidua, so that the placenta was attached directly to the myometrium. The possible causes were previous manual removal of the placenta, too vigorous uterine curettage and, in some cases, implantation on the lower uterine segment. Hysterectomy with no attempt at manual removal produced excellent results. Manual extraction of the adherent placenta was extremely dangerous.

Toxæmia of Late Pregnancy.

DR. BRENDA BRUCE SCOTT and DR. ISABEL IRELAND illustrated methods and results in early detection and management of toxæmia of late pregnancy. Statistics demonstrated reduced incidence and mortality of severe toxæmia.

Two in-patients were shown to emphasize the importance of early prophylaxis and prompt termination of pregnancy in the deteriorating case.

Histories were presented to illustrate the results of inadequate ante-natal care or failure to induce labour at a suitable stage.

Sterility.

DR. LORNA LLOYD-GREEN demonstrated the routine investigations undertaken at the Sterility Clinic.

Pregnancy Associated with a Gräfenberg Ring.

DR. RUTH WALKER showed a series of X-ray films illustrating the association of pregnancy with a Gräfenberg ring in the uterine cavity. In some cases it remained *in situ* after delivery.

Dr. Walker pointed out that, apart from any other considerations, the Gräfenberg ring was by no means a reliable means of contraception.

Pregnancy After Pneumonectomy.

DR. LESLIE WILLIAMS showed ten patients who had undergone full-term pregnancy and normal delivery after pneumonectomy for pulmonary tuberculosis. There were six *primigravidæ* and four *multigravidæ*. One patient had had a second child. None suffered from tuberculosis complications in the immediate puerperium. Nine patients were well at time of presentation, and one patient had reactivation of an old lesion six months after delivery.

Induction of Labour by Intravenous Drip Administration of "Pitocin".

DR. JUNE PASH demonstrated a method of induction of labour by means of intravenous drip administration of "Pitocin" in two patients, drawing attention to the fact that the method required constant supervision by trained nursing staff. Figures were presented illustrating the effectiveness of this means of induction, and relating the success of the method to parity, maturity and "ripeness" of the cervix.

Syphilis.

DR. JOAN DARBYSHIRE presented tables to demonstrate the decrease in incidence of syphilis over the past ten years; in particular, the number of syphilitic pregnant women had fallen from 10·1 per 1000 confinements in the year ending June, 1945, to 2·4 per 1000 in the year ending June, 1954. Attention was drawn to the need for care in interpreting the results of routine Wassermann tests, as subsequent events had shown that only a proportion of patients who have positive Wassermann reactions were syphilitic.

Two patients with latent syphilis were shown. Each woman had previously borne several syphilitic children, but after antisyphilitic treatment in the pre-natal period healthy babies were produced.

In a third case discussed, a routine Wassermann test in a pregnant woman yielded a strongly positive reaction, and the Kahn test also gave some reaction. On further investigation, the reaction was found to be caused by a recent attack of influenza, and not by syphilis.

Gynæcological Surgery in the Aged.

DR. ELLA MACKNIGHT and DR. ALISON WRIGHT presented a series of patients to illustrate gynæcological surgery in the aged. The age range of the patients was from seventy to eighty-four years. Two patients had undergone radical vulvectomy with lymphadenectomy for carcinoma of the vulva; one patient had undergone total hysterectomy and bilateral salpingo-oophorectomy for carcinoma of the body of the uterus; and several patients with *prolapsus uteri* had been treated by Manchester repair or vaginal hysterectomy with repair of the pelvic floor. Stress was placed on the importance of pre-operative preparation, expert anæsthesia and the necessity of replacement of blood as loss occurred.

Dr. Macknight also showed a patient under the joint care of Dr. Sisely and herself. The patient had presented at the hospital with a carcinoma of the lower end of the rectum infiltrating through the anus, with secondary nodules in the right *labium majus* and secondary involvement of the right superficial inguinal lymph nodes. Operation four weeks before the meeting had consisted of abdomino-perineal resection of the rectum and sigmoid *en bloc* with uterus and adnexa, vagina, vulva and inguinal lymph nodes on both sides. The iliac lymph nodes were also removed.

Diseases of the Vulva.

DR. MARGARET A. MACKIE presented a series of slides illustrating various diseases of the vulva, and discussed the diagnosis and treatment of each condition.

Congenital and Rheumatic Heart Disease.

DR. KATE MACKAY showed a series of patients with congenital and rheumatic heart disease (one of the latter complicated by subacute bacterial endocarditis) associated with pregnancy. Amongst the former were patients with Lutembacher's syndrome, interatrial septal defect, and interventricular septal defect, and two patients with coarctation of the aorta who had had successful vaginal delivery. The patient with subacute bacterial endocarditis had been successfully treated by antibiotics, with birth of a normal living child.

Treatment with Cortisone and ACTH.

DR. MARGARET HENDERSON and DR. JUNE HOWQUA demonstrated a series of patients who had been under treatment with cortisone or ACTH. They included the following: two women with rheumatoid arthritis, who had received cortisone for more than three years in dosage varying from 12·5 to 75 milligrammes daily; a woman of thirty-four years with fulminating ulcerative colitis which had failed to respond to cortisone but had remitted successfully and rapidly on intravenous administration of ACTH; a woman of forty years who, in the ninth month of her pregnancy, had developed acute *polyarteritis nodosa* due to sulphonamide sensitivity, the lesions subsiding completely and without sequelæ on treatment with cortisone; a girl of eighteen years with rheumatic fever and pericardial effusion, intolerant of salicylates and responding well to cortisone; a young woman of twenty-two years, in whom *erythema nodosum* associated with sarcoidosis was treated with cortisone; a patient with Sheehan's syndrome, in whom there had been a fair, but not dramatic, response to cortisone 12·5 to 25 milligrammes daily; two patients with acquired hæmolytic anæmia, in one of whom, a patient aged sixty-six years, the ætiology was obscure, but the red cell count was maintained at a satisfactory figure by administration of 100 milligrammes daily of cortisone, although hæmolysis was still taking place. In a woman aged forty years *lupus erythematosis* had been proven after splenectomy six years previously for thrombocytopenic purpura. The patient had a stormy course on treatment with ACTH for twelve months, developing sepsis, fluid retention and a transient psychosis. The administration of four tablets of tri-ethylene-melamine (five milligrammes) over a period of six weeks was followed by a remission, which was to date of seven months' duration; during that time no ACTH had been given.

Miliary Tuberculosis and Tuberculous Meningitis.

DR. MARTHA RENTH presented details of ten cases—four of miliary tuberculosis with tuberculous meningitis, four of miliary tuberculosis without meningitis, and two of tuberculous meningitis without miliary tuberculosis. The ages of the patients ranged from sixteen to sixty-five years, and the interval from the start of the illness from three years to six weeks. All patients are alive. All had been treated with isoniazid in varying combination with streptomycin and PAS. Intrathecal therapy was not used in three cases, was given for three weeks in two cases, and was prolonged in one case, in which also streptomycin was given intraventricularly and through a polythene tube into the basal cisterna. The last-mentioned patient was the only one who had sequelæ, in the form of atypical epilepsy.

Complications included diabetes in a patient aged sixty-five years and mitral stenosis with failed valvotomy in a patient of forty-two years; a patient aged thirty-two years developed hæmorrhagic diathesis during treatment.

At the time of the meeting seven patients were at home and three in hospital.

Blood-Stained Discharge from the Nipple.

DR. LORNA SISELY discussed the arguments for and against conservative surgery in patients presenting with a blood-stained discharge from the nipple. She said that since benign lesions were by far the commonest causes of the condition, accurate localization of the lesion with removal of duct and involved sector was advocated in preference to simple or radical mastectomy. Details of eight cases were presented (in patients from twenty-five to seventy-one years of age) in which the duct papilloma had been isolated and removed, with or without the sector (the former was considered the better procedure), with no future recurrence in the breast.

Care of the Premature Infant.

DR. KATE CAMPBELL presented a demonstration on the care of the premature infant. She divided the care into the following phases: (i) the care during birth, (ii) the care immediately after birth, (iii) the first four days, (iv) the fifth day to the ninth day, (v) the tenth day to the twenty-first day, (vi) the subsequent period.

Dr. Campbell said that during birth the emphasis was on the prevention of anoxæmia, the prevention of birth trauma, and the sucking out of the upper part of the respiratory tract as soon as the head was born from the vulva.

After birth, the usual measures were temperature control, maintenance of posture and administration of oxygen for cyanosis. Those were discussed, with the necessity for application of a sterile dressing to the cut cord. It was advised that, after twelve hours, the cord be painted with either 2·5% tincture of iodine or "Monacrin" 1:1000 in spirit, and left naked. The "Isolette" incubator was demonstrated.

Dr. Campbell said that the main danger in the first four days was atelectasis of the hyaline membrane type. The treatment of that with oxygen and "Alevaire" was shown, and the advisability of prophylactic administration of antibiotics was indicated.

From the fifth to the ninth day the main danger was severe jaundice, with the possible complication of premature kernicterus. The need was stressed to watch the urine for patchy staining of the napkins with bile. It was recommended that, in addition to the feedings, fluid be given parenterally by the subcutaneous drip method with hyaluronidase, ascorbic acid and casein hydrolysate added to the feedings. The value was discussed of exchange transfusion in the presence of deep jaundice or at the first suggestion of cerebral signs.

From the tenth to the twenty-first day the danger was that of infection, for example, bronchopneumonia.

It was suggested that ascorbic acid be given from about the fourth day, the *B* group vitamins from the fourteenth day, and the multivitamin preparation in the third week. Administration of iron was begun at the fourth week, or later if not well tolerated. The ocular fundi of premature infants of three and a half pounds or less were routinely examined at four weeks.

In consideration of the after-care, the subjects of early and late anæmia, bilateral umbilical hernia, rickets, rapid increase in skull circumference, riboflavin deficiency and malformations were discussed. It was pointed out that care in policing the administration of oxygen in the early days and weeks of the life of the premature infant, to ensure that it was given therapeutically and not prophylactically, had eliminated retrolental fibroplasia from the premature nurseries.

Purulent Meningitis.

DR. MONA BLANCH presented seven young children, who had recently recovered from purulent meningitis of varying ætiology, to illustrate the treatment of each type and emphasize the desirability of reducing intrathecal therapy to a minimum.

Of the seven cases, meningitis was due to meningococci in two, to *Escherichia coli* in two, and to pneumococci and *Hæmophilus influenzæ* in one each. In one case the ætiology could not be determined. Apart from a very slight weakness of the right side of the face and right arm and leg in the baby who had the pneumococcal infection, there were no sequelæ.

Dr. Blanch also discussed the treatment of tuberculous meningitis with streptomycin and isonicotinic acid hydrazide, and made a plea for minimal use of lumbar punctures and intrathecal therapy.

Lastly, Dr. Blanch presented a synopsis of the causes, symptoms and treatment of the so-called acute aseptic meningitis syndrome. It was illustrated by a girl of two and a half years, who had had severe vomiting for four days, followed by severe neck stiffness, and who subsequently developed a parotid swelling.

Exchange Transfusions.

DR. ELIZABETH TURNER demonstrated the method of exchange transfusion via the umbilical and saphenous veins, used at the Queen Victoria Memorial Hospital, in the treatment of infants suffering from *erythroblastosis fœtalis*.

She said that in forty-eight months, over 17,150 births had occurred at the hospital, and of the infants born 178 suffered from *erythroblastosis fœtalis*—almost 1% of all births. Added to that, 65 infants suffering from the disease had been admitted for treatment, although born outside the hospital, so that the total number of affected infants was 203. Of those, 143 had exchange transfusions; 10 had double exchange transfusions (10%). No case of kernicterus had occurred at the hospital since the practice had been established of repeating the exchange transfusion if the jaundice deepened after the first exchange transfusion; and, in spite of the fact that neurological symptoms were sometimes manifest for up to forty-eight hours in a few cases, the early signs of kernicterus appeared to be almost completely reversible after repeated exchange transfusion, as a follow-up study of the infants showed no neurological sequelæ over a period of more than eighteen months.

Dr. Turner stressed the point that the most important criterion for the use of exchange transfusion was the appearance of jaundice. She said that previously reported criteria concerning a cord hæmoglobin value of less than 14 grammes *per centum* or a positive result from the Coombs test *et cetera* were no longer tenable, as infants without jaundice tended to do well with simple transfusions and more conservative treatment in any case.

For very gross anæmia, the advisability of exchange transfusions would be considered; but the real danger in that disease lay in the development of kernicterus, and therefore the most important aim in therapy was its prevention, or reversion to normal, by treatment at an early enough stage.

The route used for the performance of repeat exchange transfusions was via the saphenous vein in the upper part of the thigh.

Intestinal Obstruction.

DR. NORRENE NICHOLSON gave an account of a series of newborn babies who had presented with the signs and symptoms of intestinal obstruction.

The first patient had begun to vomit soon after birth, the vomitus being bile-stained. Meconium was passed on the second day. The abdomen was not distended, and a straight X-ray examination of the abdomen on the second day showed gas in the bowel. Vomiting continued until the sixth day, and it was reported that one stool was passed each day. X-ray examination with barium meal was carried out on the sixth day and showed a definite hold-up at the end of the duodenum. Laparotomy was performed and revealed stenosis of the duodenum at the duodeno-jejunal junction. Gastro-jejunostomy was carried out between the antero-inferior region of the stomach and the upper end of the jejunum. The post-operative course was uneventful. The diagnosis was stenosis of the third part of the duodenum.

The second patient had begun to vomit small amounts seventeen hours after birth, the fluid being green and fæcal-smelling. No meconium was passed during the first twenty-four hours. No abdominal distension and no peristaltic waves were observed. Straight X-ray examination of the abdomen revealed distended loops of small bowel with fluid levels. Laparotomy performed after thirty-three hours revealed a double lower jejunal atresia and a volvulus. The volvulus was reduced and a jejuno-ileal anastomosis performed. There were many peritoneal bands and adhesions, which were divided. The post-operative course was stormy. The patient developed a wound infection, from which *Clostridium welchii* was grown, and he was given anti-gas-gangrene serum, penicillin and streptomycin. The diagnosis was jejunal atresia and volvulus with *Clostridium welchii* wound infection.

The third patient had begun to vomit on the third day, forceful vomiting occurring with each feed. Meconium was passed. He was admitted to the Queen Victoria Hospital on the seventh day, when he was grossly dehydrated. Straight X-ray examination of the abdomen revealed evidence of high bowel obstruction. Laparotomy was performed on the eighth day, and a volvulus of the gut was found with universal mesentery and malrotation of the gut. The bowel was untwisted and was found to be viable. The post-operative course was uneventful. The diagnosis was volvulus due to malrotation of gut and universal mesentery.

The fourth patient had begun to vomit on the sixth day, the vomitus containing mucus. The bowel actions were regular until the sixth day. Straight X-ray examination of the abdomen showed gas distributed evenly through the small bowel and no evidence of obstruction. Vomiting persisted intermittently, and on the thirteenth day laparotomy revealed an obstruction due to universal mesentery with a volvulus. The volvulus was untwisted, and the bowel was found to be viable. The post-operative course was uneventful. The

diagnosis was volvulus due to malrotation of gut and a universal mesentery.

The fifth patient had vomited intermittently from soon after birth, the vomiting becoming projectile. He was admitted to the Queen Victoria Hospital at the age of eighteen days, when he was rather dehydrated. His abdomen was distended, and some peristalsis was noted. X-ray examination with a barium bolus revealed normal stomach and duodenum but some œsophageal reflux. He was treated with "Eumydrin" and phenobarbital and intravenous administration of fluids, but vomiting continued intermittently. Stools were passed on several occasions during the period, but he was constipated. Straight X-ray examination of the abdomen at the age of twenty-six days revealed distended loops of bowel, and laparotomy was performed on the same day. The stomach and small bowel were greatly dilated. The transverse colon was also greatly dilated, the dilatation terminating abruptly at the splenic and hepatic flexures. No obstruction was found, and the abdomen was closed. The post-operative course was slow, as the patient continued to vomit intermittently for several weeks while in hospital. The vomiting persisted, in spite of medical treatment, until he was twelve months old. The diagnosis was neurogenic incoordination of the bowel.

The sixth patient had been born with an exomphalos about one and a half inches in diameter, containing small bowel. Operation was performed at the age of seven hours, and the exomphalos was found to contain loops of small bowel and a Meckel's diverticulum, which was removed. Convalescence was uneventful.

The seventh patient had begun to vomit biliary fluid soon after birth, mainly after every second feed; and when he was aged thirty-five hours, his abdomen was noticed to be grossly distended. Meconium was passed. Straight X-ray examination of the abdomen revealed distended loops of small bowel and a distended loop of large bowel. Laparotomy was performed at the age of thirty-seven hours. There was much free fluid in the peritoneal cavity. The colon was grossly distended throughout its length. Transverse colostomy was performed. The post-operative course was complicated by a wound infection. The diagnosis was Hirschsprung's disease. The patient was now aged two years and was waiting to have further investigation prior to excision of an aganglionic segment.

The eighth patient had passed no meconium during the first twenty-four hours. At the age of three days he began to vomit and had abdominal distension. That subsided, but recurred on the eighth day. At the age of seven months he was readmitted to hospital because of bouts of vomiting, constipation and abdominal distension. Straight X-ray examination revealed gross colonic dilatation but no fluid levels to suggest obstruction. Examination with barium clysma confirmed the presence of megacolon, but no narrow segment was present. X-ray examination at twelve months showed the calibre of the lower part of the sigmoid colon to be less than the rest of the colon. X-ray examination at seventeen months showed a definite narrow segment in the lower part of the sigmoid. The patient was now awaiting operation for resection of the aganglionic segment. The diagnosis was Hirschsprung's disease.

Neonatal Lung Conditions.

DR. GWEN HEWITT presented material relating to neonatal lung conditions. X-ray films were shown demonstrating atelectasis in the newborn period, including a case in which the patient was diagnosed as having hyaline membrane and recovered. In that case, the baby's condition was satisfactory at birth, and breathing was established almost immediately. Cyanosis with poor air entry, grunting respirations and bilateral rib retraction developed at six hours, and at thirty hours the X-ray examination showed fine nodulation in both lungs, consistent with a diagnosis of hyaline membrane formation.

Complications illustrated included mediastinal emphysema and tension pneumothorax. In the example given of the latter condition the baby had been born by Cæsarean section for *placenta prævia*. The infant was slow to breathe and required resuscitation. The respiratory rate rose, and examination showed gross rib retraction and poor air entry on the right, with depression of the liver to three inches below the right costal margin. Air was removed from the right side of the chest, and the infant gradually recovered.

The difficulties of differential diagnosis were illustrated by a case of diaphragmatic hernia. A premature baby had done well until the sixth day, when it became cyanosed and breathless. Examination revealed rib retraction and feeble breath sounds. The baby's condition was thought to be too poor for it to stand X-ray examination at first. After administration of oxygen, antibiotics and fluids by the subcutaneous route, the condition improved, and X-ray examination showed abdominal contents in the chest. The baby made an excellent recovery after operation.

Microscope slides were used to illustrate the appearance of a normal fœtal lung and the results of inhalation of *liquor amnii*, intrauterine pneumonia and hyaline membrane formation.

Ante-Natal Blood-Grouping Tests.

DR. ELSIE ABRAHAMS and DR. GERTRUDE HILLER demonstrated the routine ante-natal blood-grouping tests, including investigation of A B O incompatibility.

Radiology of the Œsophagus.

DR. J. HAMILTON SMITH discussed the radiology of the œsophagus. The subject of acute and chronic œsophagitis was outlined; the predisposing factors, the association with hiatus hernia in most cases, the pathology, the clinical picture and the radiological signs of the condition and of its complications were presented. The characteristic features of carcinoma of the œsophagus were illustrated by reference to selected radiographs. Similarly, cardiospasm was discussed and illustrated; the incidence in males as well as in females was noted, also the occurrence of the condition occasionally in the very young and very old. The significance was given of recurrent and chronic pulmonary infections in those cases.

Finally, radiographs were present demonstrating less common diseases of the œsophagus; they included diverticuli, œsophageal varices, a "ripple œsophagus" and the "post-cricoid" webs of the Plummer-Vinson syndrome.

Radiology of the Biliary Passages.

DR. JOAN SPONG discussed the radiological demonstration of the biliary passages, with particular reference to intravenous cholangiography, with "Biligrafin" and organic iodine compound of low toxicity. She said that with normal liver function, the bile ducts, as well as the gall-bladder, were visualized, and the bile ducts could be investigated after cholecystectomy. Oral administration of "Telepaque" remained the method of choice for routine use, but the intravenous route should be used in the investigation of the "post-cholecystectomy syndrome", in cases of non-vizualization of the gall-bladder by oral methods, possibly pre-operatively, if the presence of common bile duct calculi was suspected. Contraindications were obstructive jaundice, severe hepatic dysfunction and iodine sensitivity.

A series of X-ray films were shown. The first was from a case of cholecyst-duodenal fistula, in which barium entered the common bile duct during a barium meal. Next was a group of films providing examples of oral cholecystography. Finally were films showing intravenous cholangiography, including those from cases of common bile and cystic duct stones. One patient had suffered severe recurrent attacks of typical biliary colic for five years after cholecystectomy. A large stone in the common bile duct was demonstrated. She had been free from symptoms since its removal.

Demonstrations of Tonsillectomy and Anæsthesia.

DR. H. E. FINNEY performed three tonsillectomy operations. DR. JOAN CROSIER administered the anæsthetics. The first patient was a child aged three years, the second a girl of twelve years, and the third a woman of twenty-four years.

The two children were premedicated with "Seconal", one and a half grains two hours before operation, and atropine, one one-hundred-and-fiftieth of a grain one hour before operation. The anæsthesia was induced with ethyl chloride and open ether. The older child had a "sleeping" dose of "Pentothal" before the ethyl chloride. Anæsthesia was maintained with insufflated ether from a blow-over machine to the Boyle Davis gag. The adenoids were first removed in both cases and the tonsils dissected out. One catgut tie was used on the younger child to control persistent bleeding from the upper pole plexus.

The adult was premedicated with "Nembutal", one and a half grains two hours before operation, and morphine, one-sixth of a grain with hyoscine, one one-hundred-and-fiftieth of a grain one hour before operation. Induction of anæsthesia was with 0·5 grain of "Pentothal"; then 75 milligrammes of "Scoline" were given, and the patient was intubated through the nose, direct laryngoscopy being used. Anæsthesia was maintained with endotracheal "Trilene" and oxygen from a blow-over type of machine. The tonsils were dissected out.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the New Medical School, Frome Road, Adelaide, on April 1, 1955.

Aspects of Virus Multiplication.

PROFESSOR P. M. DEBURGH read a paper on some aspects of virus multiplication. He said that the use of large infecting doses of ectromelia virus, of the order of 10^8L.D.$_{50}$ given intravenously, enabled nearly all liver cells of the mouse to be infected at one time. That provided a convenient model for the study of the multiplication of the virus. The growth curve showed a drop to about 1% of the initial level followed by a rise to the initial level at twelve hours, where it remained until about eighteen hours when a thirty-fold increase occurred, followed by a second rise some hours later, also of about thirty-fold. As practically all cells were infected initially, those rises represented the events taking place in each cell.

The liver cells in which the virus growth occurred showed changes. The amount of cytoplasm increased more rapidly than the nucleus, and there was an increase in protein and fat as well as a small increase in ribonucleic acid, but no increase in desoxyribonucleic acid, and no increase in the number of cells. The increase in cell size (which was well marked twenty-four to thirty hours after infection, necrosis of cells becoming evident at thirty-six to forty hours) was not due to virus material, which accounted for one-twentieth or less of the increase in cell substance.

An early change in function found in this system was the inability of infected cells to form glycogen which appeared about sixteen to eighteen hours after infection. Until late in infection there was little change in oxygen consumption, fructose uptake or lactic acid production.

Infection of livers regenerating after partial hepatectomy had also been studied. Virus grew somewhat faster in regenerating liver and produced cell damage earlier. A most interesting effect of virus growth in regenerating liver was the inhibition of mitosis produced by the early stages of virus infection. At about forty hours after partial hepatectomy in young mice, a mitosis rate of about 7% was found. If virus was injected twelve hours before that, the mitosis rate was reduced to 1·5%. That effect was produced before there was any histological change and before any actual multiplication of virus or detectable increase in soluble virus antigen. The effect was not due to interference with synthesis of DNA. It was considered that the control of mitosis might be a demonstration of the gene-like action of a virus in taking control of a cell.

Some Aspects of Poliomyelitis.

DR. R. V. SOUTHCOTT discussed some aspects of poliomyelitis. He briefly outlined the history of poliomyelitis from the 1770's to the present day. He said that whereas Sir Walter Scott's illness in 1773 was probably poliomyelitis, suggestions of earlier occurrences of the disease, based on ancient carvings, paintings, skeletons *et cetera*, were purely speculative. Stress was laid on the changes in the features of the disease and the size of epidemics. The epidemiological features were outlined, and reference was made to the two chief schools in regard to the mode of spread of the disease: (i) droplet borne, (ii) enteric. Those were discussed. It was suggested that it was possible that the mode of spread of the disease in non-epidemic times differed from that occurring during epidemics. Detailed study of the mode of spread suggested that during epidemics it was by contact spread by droplets. Reference was made to the finding during the 1947-1948 South Australian epidemic of approximately two non-paralytic cases to each paralytic case, and the implications of that were discussed. Snow's recent account of the poliomyelitis epidemic in Western Australia at the time of the Queen's visit was referred to. Dr. Southcott regretted that more use had not been made of that epidemic in following up the epidemiology. The precautions used appeared to have been based largely on the assumption that the spread of the disease was by enteric means—for example, the stress laid on the possibilities of spread by fingers. He considered it would have been legitimate for the public health authorities to have issued, optionally, a light plastic mask to shield at least the nose and mouth from droplet spray. That would have been inexpensive, and data might have been obtained enabling a comparison to be made between the different possibilities of spread.

Predisposing factors were discussed, including racial and social, nutritional and immunological factors. The possible mechanism of poliomyelitis following inoculations was considered. Discussing the relationship between poliomyelitis and tonsillectomy Dr. Southcott said that he believed that in such bulbar cases the portal of entry of virus was via the motor pathway to the *nucleus ambiguus* to the pharynx. Stress was laid on the value of the work of Baker and his colleagues in outlining human respiratory and circulatory centres in the *medulla oblongata* from a study of bulbar cases. Dr. Southcott submitted the possibility of preventing the predisposing effect of tonsillectomy (long range) to the contraction of bulbar poliomyelitis by the Emerson type of tonsillectomy operation. In that operation the tonsillar fossa was, by a plastic procedure, left covered with normal pharyngeal epithelium, thereby possibly denying poliomyelitis virus access to the neurones to the pharyngeal musculature.

A MEETING of the Medical Sciences Club of South Australia was held in the New Medical School, Frome Road, Adelaide, on May 6, 1955.

Symposium on Radiation Effects.

A symposium was held on radiation effects.

Genetic Considerations.

PROFESSOR D. G. CATCHESIDE said that there was an underlying relationship in the causation of genetic changes, expressed in descendants, and of injury, such as blood damage, tumours, leuchæmias, caused to an exposed individual. All involved destruction or loss of function of various constituents of cells. Non-genetic material destroyed might be replaced, after a period of depression of metabolic activity. But after such a shock, the cell might be altered in its mode of differentiation, including rate of division; that might be the way neoplasms were induced.

The killing of cells was usually consequent upon injury to the chromosomes, either by producing "sticky" mitoses or by producing breaks in them proportional in number to the dosage received. Loss of the acentric fragments and disorganization of mitosis by the production of sister union or interchange chromosome bridges conspired to the death of cells and tissues. Tissues showed "recovery" through uninjured or less injured cells replacing the damaged and killed ones.

Finally, the genes might be altered or minute deletions of parts of the chromosomes produced, the latter simulating gene mutations, often in a more extreme form. The number of such genetic changes was linearly proportional to dosage and independent of dose rate down to the lowest tested. They appeared to be strictly cumulative and to show no recovery towards the normal. The genetic changes as a whole were harmful. A population subjected to radiation hazards would have those induced changes added to the already heavy load of harmful genes that it carried. That would result in a further lowering of the average fitness of the population, at a rate dependent upon the dosage received by the population. Calculations of the possible effect might be made upon the basis that, as in other organisms such as flies and mice, a dose of 100*r* to immature germ cells induced as many new mutations as appeared spontaneously in the lifetime of an individual.

Clinical Effects.

PROFESSOR H. N. ROBSON presented a brief account of the clinical effects on human beings of ionizing radiations. He said that information on that subject had, of course, been amassed from multifarious sources, of varying degrees of reliability. The relative sensitivity of the various tissues of the body was fairly accurately known, the most sensitive being lymphocytes and lymphoid tissue, followed by myeloid cells, epithelial structures, endothelium, connective tissues including muscle and bone, and nervous tissue in that order.

The main types of ionizing agent could roughly be divided into two groups, with X rays, γ rays and neutrons as the main external hazards, while β and α particles were poorly penetrative, and therefore the main danger from those was by ingestion or inhalation.

Clinical effects were then described, under the main headings of those resulting from external and those from internal irradiation. The minimal lethal dose for human beings was

thought to be about 400r of external irradiation. Acute effects included the syndrome of irradiation sickness, the details of damage to hæmatopoiesis, skin and gastro-intestinal tract later manifesting themselves at varying intervals. Chronic external exposure (maximum tolerance of the order of 0·1r per week) had resulted in aplastic states of bone marrow, possibly leuchæmia, sterility and chronic states in bone and skin, sometimes neoplastic. Internal irradiation produced similar changes, both acute and chronic, the results being, of course, modified by the site of deposition in the body of the radioactive material, the quantity and the half-life of the isotope.

Physical Features.

MR. B. W. WORTHLEY said that the ionizing particles which caused biological effects in living tissues were photons (X and γ rays), fast electrons, fast protons and neutrons. Biological response must be correlated with "dosage" of radiation in appropriate units. The fundamental unit was that of energy absorbed in ergs per gramme of tissue. The practical unit was the röntgen, and was measured by the estimation of electrical charge released by the passage of radiation through an air cavity one cubic centimetre in volume in the material. That unit had been replaced by the röntgen-equivalent-physical (R.E.P.). One gramme of air irradiated with one röntgen absorbed 83 ergs. Dividing the energy expressed in ergs absorbed in one gramme of tissue by 83 ergs gave the dose in reps. The distinction was important when bone was encountered, as the dose expressed in reps might be as much as ten times that expressed in röntgens. For water-equivalent materials the difference was usually about 10%. The röntgen-equivalent-mammal (R.E.M.) was another unit related to the rep via the relative-biological-effectiveness (R.B.E.). The R.B.E. was an empirical number which assessed the efficiency of a given type of radiation in producing a given biological effect when compared with an equal dose in röntgens of the γ rays from radium. The dose in rems was given by the product of the dose in reps and R.B.E. Natural sources of radiation included cosmic rays, γ rays from radium in the soil and building material, drinking water and radioactive potassium in food. Those gave approximately one two-hundredth of the permissible dose of 500 milliröntgens per week. Some sources of hazard were various X-ray machines, radioactive isotopes and even the television screen.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

SANITATION IN SYDNEY.

[From the *New South Wales Medical Gazette*, December, 1871.][1]

THE presence of several cases of English cholera and colliquative diarrhœa, lately, in the metropolis, is no doubt attributable to a great extent to the advent of the summer season, and may be occasioned in some instances by the consumption of unripe fruit, the presence of which in the intestinal canal occasions disturbance in the chylopoietic viscera. Although these diseases partly originate from the abovementioned causes, yet we have reason to believe that the absence of drainage in a great part of the city, the system of cesspools that prevails in the suburbs and a portion of the city, the presence of cowyards in the crowded lanes of Woolloomooloo, and the meaner portions of the town, together with the contaminated state of the water of Port Jackson, encourage and increase the liability to many diseases of a Zymotic character. We know of many instances in which old cesspools have been allowed to overflow for years without having been visited once during this time by the night cart. The disgusting effluvium of these reeking abominations pervades the surrounding atmosphere, while their contents are allowed to run into the open, and contaminate the open drains, causing injury to the health of the citizens in the same way as does an illdrained camp to an army, or the badly ventilated wards of a hospital to the poor patients compelled to reside within them for the treatment of their numerous ailments. It would require a Hercules to clean out this Augean Stable. In England, fifteen years ago, when Asiatic cholera raged, a cry of warning went up through the length and breadth of the land, and in consequence almost every house was provided with a cistern of water and a pan, which communicated by a pipe with a closed drain, by means of which the refuse matter was carried off. The question forces itself upon us, shall we rest quiescent in our dirt and disagreeable smells like the people of the East? Wait like England, until the cholera is present among us, before we attempt a proper system of drainage? Or, rather, profiting by the history of previous epidemics, that have happened in other lands, at once take measures to make Sydney the sweetest and healthiest, as it is by nature the fairest city in the Southern Hemisphere.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

RESTORATION OF DRUG AUTHORITY.

SIR: I desire to inform you that in accordance with the provisions of Regulation 25 (2) under the *Police Offences (Amendment) Act*, the Minister has directed that the withdrawal of the authority of Doctor Patrick Howard Speight, of 12 Hassall Street, Parramatta, to procure and be in possession of drugs to which the Act applies for the purpose of his profession and to issue prescriptions for such drugs, shall cease to operate on and from Friday, August 19, 1955. Would you be good enough to publicize the restoration of this authority in your journal?

Yours, etc.,

E. J. BUTTSWORTH,
Under Secretary.

Chief Secretary's Department,
Sydney,
August 15, 1955.

THE BAN ON HEROIN.

SIR: The lay Press has recently given some prominence to the banning of the drug heroin. Whether there were a significant number of heroin addicts in Australia was one of the points raised. Unless this addiction is a public health menace, I think the profession should be more fully consulted before such a valuable drug is denied us.

Most practitioners would agree that 30% to 40% of patients given morphine have vomiting and prostration following its use, and in the case of a shocked patient with myocardial infarction the prognosis must surely be worsened. Substitutes for morphine such as "Amidone" and "Pethidine" are just as bad in this respect or are ineffective. Methylmorphinan ("Dromoran") is effective, but is a restricted drug in the National Health Scheme and also causes vomiting occasionally.

Although the evils of heroin addiction are great, I feel we should be given proof that as a profession we are not to be entrusted with its use. It would seem that the banning of heroin is an expedient originating in the higher echelons of administrative medicine rather than a necessity urged on by abuse of the drug at a practitioner level, and as one who has found the drug a valuable "second string" I register my protest against its prohibition.

Yours, etc.,

Z. FREEMAN.

217 Macquarie Street,
Sydney,
August 14, 1955.

Post-Graduate Work.

THE MELBOURNE MEDICAL POST-GRADUATE COMMITTEE.

PROGRAMME FOR SEPTEMBER, 1955.

Courses by Overseas Lecturers.

PROFESSOR D. R. MACCALMAN, Nuffield Professor of Psychiatry, University of Leeds, will deliver the following lectures at 8.15 p.m. in the Medical Society Hall: Thursday, September 8, "The Impact of Psychiatry on General

Medicine"; Monday, September 12, "The Early Symptoms of Mental Illness".

Sir Stanford Cade, surgeon and radiotherapist, of London, will speak on "Recent Advances in the Treatment of Cancer" at 8.15 p.m. on Thursday, September 15, in the Medical Society Hall.

Professor Brian Windeyer, of the Myerstein Institute of Radiotherapy, Middlesex Hospital, London, will accompany Sir Stanford Cade on his visit to Melbourne. Professor Windeyer will lecture for specialists on "Super-Voltage Therapy" at 8 p.m. on Tuesday, September 13, at the Cancer Institute.

Dr. W. S. C. Copeman, Chairman of the Empire Rheumatism Council, London, will deliver the following lectures at the Medical Society Hall at 8.15 p.m.: Monday, September 19, "The Differential Diagnosis of Arthritis"; Thursday, September 22, "The Rheumatic and Pararheumatic Collagen Diseases".

Attention is drawn to the following lecture, arranged by the Royal Australasian College of Surgeons, which will be open to all members of the medical profession: Professor R. I. Harris, Sims Commonwealth Travelling Professor, will lecture at the College at 8.15 p.m. on Friday, September 9, on "Tuberculosis of Bones and Joints".

Fees will be at the rate of 15s. for the lectures by Professor MacCalman, Sir Stanford Cade and Dr. Copeman, but those who have paid an annual subscription to the Post-Graduate Committee may attend without further charge.

Country Courses.

Warrnambool.

On September 10, at Warrnambool, Dr. Keith D. Fairley will lecture on "Thyrotoxicosis". A second lecture is to be arranged.

Mildura.

On September 17 and 18, at Mildura, the following lectures will be given:

Saturday: 2.30 p.m., Dr. W. J. Rawlings, "Habitual Abortion"; 4 p.m., Dr. G. R. A. Syme, "Thyroid Disease".

Sunday: 8 a.m., Dr. G. R. A. Syme, "Thyroidectomy"; 9.30 a.m., Dr. Pincus Taft, "Endocrine Therapy"; 11 a.m., Dr. Norman Wettenhall, "Failure to Thrive in the Child".

Flinders Naval Depot.

On September 14, at 2.30 p.m., a demonstration will be given at *Flinders Naval Depot by Dr. Keith Bradley on* "Neurosurgical Conditions of the Neck and Upper Limbs". This is by arrangement with the Royal Australian Navy.

Refresher Courses for General Practitioners.

Gynæcology and Obstetrics.

From September 26 to October 7 a full-time course in gynæcology and obstetrics at the Royal Women's Hospital will be conducted by members of the honorary staff for the Melbourne Medical Post-Graduate Committee. The fee for this course is £12 12s.

Medicine and Surgery.

A general course in medicine and surgery will be held from October 10 to 14 inclusive. This will be a full-time course, classes commencing each day at 9.30 a.m. and continuing until 5 p.m., at the following places: Monday, the Royal Melbourne Hospital; Tuesday, the Alfred Hospital; Wednesday, Prince Henry's Hospital; Thursday, Saint Vincent's Hospital; Friday morning, Fairfield Hospital; Friday afternoon, the Eye and Ear Hospital. The fee for this course is £7 7s. A detailed timetable will be published shortly. The Post-Graduate Committee will conduct the course.

Royal College of Obstetricians and Gynæcologists.

On September 18 the Victorian State Committee of the Royal College of Obstetricians and Gynæcologists will conduct an all-day meeting at the College House, 8 Latrobe Street, Melbourne, from 11 a.m. till 4 p.m. The fee for the meeting, including dinner, will be £2, payable to the Secretary of the College. Details of the programme have already been published.

Neuropathology for D.P.M. Part II.

Lectures in neuropathology for D.P.M. Part II will be conducted at the Alfred Hospital during September.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED AUGUST 13, 1955.[1]

Disease.	New South Wales.	Victoria.	Queensland	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	3(1)	3(1)	4(1)	..	..	..	..	..	10
Amœbiasis	..	..	1(1)	..	..	..	..	..	1
Ancylostomiasis	1	..	10	..	..	..	..	..	11
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	5(4)	6(5)	4(4)	..	1	1	..	..	17
Diphtheria	3(1)	3(3)	1	..	7(7)	..	..	..	14
Dysentery (Bacillary)	..	1(1)	1(1)	..	..	1	..	..	3
Encephalitis	..	..	..	2(1)	..	..	..	..	2
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	..	..	..	..	1(1)	..	..	1
Infective Hepatitis	51(18)	72(34)	..	7(6)	3(1)	3	..	..	136
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	2	..	2	..	1	..	5
Leptospirosis	..	..	34(1)	..	..	..	..	..	34
Malaria	..	1	1	..	..	..	4	..	6
Meningococcal Infection	4(3)	4(4)	1(1)	..	..	2(2)	..	..	11
Ophthalmia	..	..	..	..	2(2)	..	..	..	2
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	1	3(1)	1	2(2)	..	..	..	..	7
Puerperal Fever	..	..	..	..	1	..	..	..	1
Rubella	..	22(11)	..	6(1)	2(1)	..	..	..	30
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	9(6)	12(7)	33(11)	9(6)	1(1)	2(1)	..	..	66
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	..	..	1	..	2
Trachoma	..	..	..	..	4(1)	..	..	..	4
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	37(34)	19(11)	15	9(4)	4(2)	4(2)	3	..	91
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

General.

The address of the Melbourne Medical Post-Graduate Committee is 394 Albert Street, East Melbourne. Telephone: FB 2547.

Research.

THE AUSTRALIAN NATIONAL UNIVERSITY: THE JOHN CURTIN SCHOOL OF MEDICAL RESEARCH.

APPLICATIONS are invited for scholarships in the departments of biochemistry, microbiology, physiology and experimental pathology of the John Curtin School of Medical Research at the Australian National University, Canberra, A.C.T. Work is being carried out in these departments on phosphate metabolism, pox and influenza viruses, neurophysiology and tuberculosis respectively.

Applicants should have a master's degree or equivalent research experience. Scholars are expected to enrol for a Ph.D. degree, and will normally be expected to commence their studies in March, 1956. The scholarships are tenable for two years and may be renewed for a third year.

Allowances are as follows: £A705 *per annum* if single; additional allowance normally granted to a married scholar with children; grant towards scholar's travel to Canberra.

Application forms and other particulars are available from the Acting Registrar, Australian National University, Box 4, G.P.O., Canberra, A.C.T., to whom applications should be submitted by October 15, 1955. It is hoped to announce the awards early in December.

Notice.

BEATTIE-SMITH MEMORIAL LECTURES.

THE twenty-first Beattie-Smith Memorial Lectures on Insanity will be delivered by Dr. Ainslie Meares in the Anatomy Lecture Theatre, University of Melbourne, at 8.15 p.m. on Monday, September 26, and Monday, October 3, 1955. The subject of the lectures will be "Recent Work in Hypnosis and its Relation to General Psychiatry". The lectures are open to all members of the medical profession, and entry is free and without ticket.

Nominations and Elections.

THE undermentioned has been elected as a member of the South Australian Branch of the British Medical Association: Poynton, John Orde, qualified 1930, London.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Brookes, Ronald Alfred, M.B., B.S., 1955 (Univ. Sydney); Davies, Howard Edwin, M.B., B.S., 1955 (Univ. Sydney); Swan, Lorelle Hesperie, M.B., B.S., 1955 (Univ. Sydney); Bowie, Marion Louie, M.B., B.S., 1954 (Univ. Sydney); Roberts, John, M.B., B.S., 1954 (Univ. Sydney); McGovern, Kenneth Francis, M.B., B.S., 1953 (Univ. Sydney); Hanrahan, Edmund Loftus Bede, M.B., B.S., 1940 (Univ. Sydney); Briedis, Antonina, registered in accordance with the provisions of Section 17 (1) (*c*) of the *Medical Practitioners Act*, 1938-1955.

Deaths.

THE following deaths have been announced:

CALDWELL.—Wallace Edwin Caldwell, on August 10, 1955, at West Perth, Western Australia.

HALLION.—Basil John Hallion, on August 12, 1955, at Fremantle, Western Australia.

MORRIS.—Matthew James Morris, on August 14, 1955, at Dalkeith, Western Australia.

TOWNSEND.—Ruby Adele Townsend, on August 14, 1955, at Melbourne.

BROOKES.—George Arthur Brookes, on August 16, 1955, at Sydney.

BRIERLEY.—Frank Arthur Brierley, on August 20, 1955, at Sydney.

Diary for the Month.

SEPT. 3.—Queensland Branch, B.M.A.: Annual Meeting.
SEPT. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPT. 7.—Victorian Branch, B.M.A.: Clinical Meeting.
SEPT. 7.—Western Australian Branch, B.M.A.: Branch Council.
SEPT. 9.—Tasmanian Branch, B.M.A.: Branch Council.
SEPT. 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
SEPT. 19.—Victorian Branch, B.M.A.: Finance Subcommittee.
SEPT. 20.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 21.—Western Australian Branch, B.M.A.: General Meeting.
SEPT. 21.—Victorian Branch, B.M.A.: Clinical Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all contract practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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